Genetic Evidence of Sex Difference in Autism Spectrum Disorder (ASD): A Review

By

Name: Sumaia Akter Muna

ID: 18146103

A thesis submitted to the Department of Pharmacy in partial fulfilment of requirements for the degree of Bachelor of Pharmacy (Hons)

School of Pharmacy

Brac University

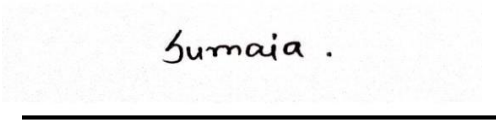
February 2022

Declaration

It is hereby declared that

- The thesis submitted is my own original work while completing degree at Brac University.
- The thesis does not contain material previously published or written by a third party, except where this is appropriately cited through full and accurate referencing.
- The thesis does not contain material which has been accepted, or submitted, for any other degree or diploma at a university or other institution.
- I have acknowledged all main sources of help.

Student's Full Name & Signature:



Sumaia Akter Muna

18146103

Approval

The thesis titled "Genetic Evidence of Sex Difference in Autism Spectrum Disorder (ASD): A Review" submitted by Sumaia Akter Muna (18146103) of Summer 2021 has been accepted as satisfactory in partial fulfillment of the requirement for the degree of Bachelors of Pharmacy.

Examining Committee:

Supervisor:

(Member)

Dr. Sharmind Neelotpol
Associate Professor, School of Pharmacy
Brac University.

Deputy-Chair:

(Member)

Dr. Hasina Yasmin
Professor & Deputy-Chair, School of Pharmacy
Brac University.

Dean:

(Chair)

Dr. Eva Rahman Kabir
Professor and Dean, School of Pharmacy
Brac University.

Ethics Statement

This study does not involve any human and animal trial.

Abstract:

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition that has prominent genetic evidence of having gender difference in the identification and distribution of ASD characteristics in patients. The aim of the review is to emphasize on the genetic evidence of gender difference in ASD. This study assists in acknowledging the major reasons behind males being prone to be diagnosed or affected with this disorder. Here, the initial cause was mutations and neuron dysfunctions followed by the ASD risk genes and its association with lncRNAs. Then, the impact of sex difference biology was grounded in. Lastly, the significance of RORA receptor regulation predicted male biased prevalence in ASD was described in the review. Nevertheless, profound future work from both ASD and the genetic discussion of the gender difference in ASD is a subject to look forward to enrich our understanding on the mechanism of the noted factors in attuning ASD risk.

Keywords:

Autism Spectrum disorder, sex difference, genetic evidence, prevalence, female protective bands, testosterone, estrogen, estradiol, sex hormones, neurodevelopmental, dysfunction, mutation.

Dedication:

Dedicated to my parents, friends and my project supervisor, Dr. Sharmind Neelotpol.

Acknowledgements

To begin with I would like to thank Almighty for His unlimited blessings in attempt to empower me with the strength and willingness to accomplish this project work.

I would like to express my sincere gratitude to my project and academic supervisor, Dr. Sharmind Neelotpol (Associate Professor, School of Pharmacy, Brac University) for her valuable supervision and enthusiasm throughout this project. She was genuinely a source of advice and support throughout my study and project writing. I am incredibly obliged for her precious feedback and suggestions throughout my research that helped me a lot to complete this project work smoothly.

I would also like to express my humble gratitude to Dr. Eva Rahman Kabir (Professor and Chairperson, School of Pharmacy, Brac University) for her dedication, contribution and guidance towards the student as well as the department.

Finally, I would like to express my gratitude to my parents who keep inspiring me to go beyond my limits. I would not have come this far without their constant prayers and unconditional love. I would also like to thank all the people who, whenever needed, have helped me with their utmost abilities.

Table of Contents

List of tables

List of Acronyms

ASD	Autism Spectrum Disorder
PDD-NOS	Pervasive Developmental Disorder-Not Otherwise Specified
lncRNAs	Long non-coding RNAs
RORA	RAR- related orphan receptor alpha
ER	Estrogen
AR	Androgen
DHT	Dihydrotestosterone

Chapter 1:

Introduction:

Autism spectrum disorders (ASD) are a group of complex neurodevelopmental abnormalities defined by difficulties in social communication and limitations in activities and interests (Zhang et al., 2020). ASD is linked to a number of characteristics, including apprehension in social situations, unusual interest in objects, agitation in response to changes in the environment, under or over reaction to the five senses (sight, touch, taste, smell, hearing), repetitive movements, and complicated emotions (Williams & Williams, 2019).

ASD begins mostly around approximately the age of 3 years and can last throughout the lifetime whereas some symptoms improve and others stay unchanged. ASD diagnosis shows different conditions that are used separately; autistic disorder, pervasive development disorder (PDD-NOS) and lastly Asperger's syndrome. All these conditions are now being called as autism spectrum disorder. Other characteristics of this disease can include, delay in language skills, movement skills, epilepsy or seizure disorder, anxiety, stress, gastrointestinal issues etc. (National Center on Birth Defects and Developmental Disabilities, 2021).

In passing years, epidomiological studies showed rapid increase in the prevalence of ASD. It is reported that throughout the world, 1 out if 150 children are victim of ASD. The frequency of autism in South Asia varies from 0.09 percent in India to 1.07 percent in Sri Lanka, according to a comprehensive investigation. The accuracy of ASD prevalence statistics is determined by diagnosis criteria, age, geographic region, service availability, and public awareness. Among the many risk variables, advanced maternal and perinatal age is one of the most notable, with a 10-year rise in maternal age greatly increasing risk. Autism is under-reported in Bangladesh, with an

incidence of 0.15-0.8 percent according to a research. Bangladesh's Ministry of Social Welfare conducted a significant study in 2016 showed that autism was conquering about 19% of total neurological disabilities recorded (Akhter et al., 2018).

ASD is accompanied several complications such as eczema, skin allergies, frequent infections, gastrointestinal complications. These patients also suffer from social deficit, language impairment, repetitive behaviors, brain dysfunction along with other neurological disorders, being prone to tumors etc. followed by other problems hindering one's daily life (Al-Beltagi, 2021).

Furthermore, because males are more likely to be diagnosed with ASD, this illness is often detected by severe sexual dimorphism, and this information is crucial in determining the underlying etiology. Many explanations have been offered to explain why guys are at an increased risk of ASD. The theory of multiple threshold liability, which hypothesizes that several genetic factors contribute to the risk of developing ASD and that a higher threshold of genetic liability is required for females than for males, is known as the "female protective model" among the theories. Female instances contain an excessive detrimental copy number of variations, which are responsible for disruption of more genes than those observed in males, according to the hypothesis. Similarly, the "extreme male brain theory," which highlights gender bias beliefs and posits that fetal testosterone exposure may underpin gender differences in autistic symptoms, is a widely discussed popular notion. A number of studies led to the trip by presenting evidence in support of this notion. In certain investigations, steroidogenic activities were found to be enhanced during fetal development in boys with ASD, but females exposed to high levels of testosterone in the womb were born with a male-typical play style. When compared to male ASD patients, female ASD patients had a higher probability of low intellectual level and more internalizing symptoms. Male patients, on the other hand, displayed more social and externalizing behavioral difficulties, such as aggressive actions

and an increase in stereotyped behaviors. Another structural neuroimaging investigation found specific frontal lobe anomalies in solely male patients, as well as substantial gender differences in the motor system and "social brain" areas. Again, androgens had a prenatal male-biased influence on the circularity of this particular brain area (Zhang et al., 2020).

The study of genetic evidence in ASD is important as it helps in the identification of diagnosis as it works as a bridge between medical and scientific community to sum up the risk factors. ASD is currently thought to be a condition resulting from a complex combination of genetics and environment, with heritability ranging from 40 to 80 percent. Environmental risk factors have been elucidated through epidemiological studies, but there is still much to learn about how they interact with genetic predisposition to contribute to ASD etiology. Individuals with similar pathogenic mutations can have substantially different phenotypes in complicated disorders. Individuals with the same variations occurring on opposite ends of the spectrum have genetic modifier factors that modulate the expression of other genes. This study will significantly disclose about the genetic landscape of ASD, copy number variant, de novo mutations, epigenetic influence, sex linked effects and many more aspects (Rylaarsdam & Guemez-Gamboa, 2019)

While there is a lot of interest in ASD in the scientific world right now, and significant progress has been achieved in numerous areas of research, the underlying pathophysiology of this study is still unknown. Given that ASD is thought to be the most heritable of all developmental neuropsychiatric disorders, the discovery of susceptibility genes could pave the way for a better understanding of the disease's underlying cellular and molecular mechanisms, as well as improved diagnosis and the development of novel therapeutic strategies. Despite the fact that there is solid genetic evidence, the rate of progress is not satisfactory. There's still a chance that the link between genetic research and social incapacity hasn't been fully established. The assuring part is highly

effective advocacy groups, rapidly evaluated genomic tools and upgraded methodologies with development of gene collection have been showing promising improvement in discoveries over the next several years and so more prominent methods should be adapted in order to prove the significance of genetic evidence in ASD (Verghese, 2011).

Nevertheless, whereas more than 100 ASD susceptibility genes have been discovered, genetic specific mutations are found in only about 8% of patients. Although mutations account for just a tiny percentage of autism cases and genetic identification is not yet attainable for the majority of ASD patients, the information about genome function offered by transcriptome data is critical for further research. These data have proven to be particularly useful in comparing groups of people with ASD to control samples in order to determine gene dysregulation.

The study also important in measure the protein levels with analyzing genetic information, highlights the dysfunction of brain, immune system, cell metabolism and embryology that assist in diagnosis of ASD (Ansel et al., 2017).

To sum up, the study on genetic evidence of gender difference in ASD is promisingly focusing on assisting to show the significance of genetic information in diagnosis of ASD. This will help in underlying the differences in symptoms, adaption and diagnosis capacity with the changes of phenotypes and genotypes. Lastly, this study also can work in showing the ground where males are more prone to have ASD Candidate genes than females. The sex chromosomes explain this mechanism that will help with the further procedure to deal with ASD.

Aim and Objectives:

The aim of the study was to find out the genetic evidence of sex difference in ASD.

Objective of the study:

The objectives of the study were-

- to detect the perspective of analysis at the co expressions of genes that highlights the dysfunction in neurological, metabolic and other body mechanisms.
- to demonstrate the sex specific genes and their noteworthiness in ASD.
- to show the ground for males being more prone to have ASD than females.

Chapter 2:

Methodology:

This review was based on recent and relevant research papers and articles from journals with a high impact factor. A thorough search was conducted in peer-reviewed publications, official papers, and articles. Basic and supplementary knowledge was gathered from books to supplement the review effort. ResearchGate, Google Scholar, ScienceDirect, PubMed, NCBI, and other search engines were utilized to gather data for this review. To have an optimal quality evaluation on the significance of genetic evidence of gender difference in ASD, a thorough search of the sources was conducted, followed by a narrowing down to the most recent and relevant ones.

Chapter 3:

Findings and Discussion:

After evaluating the significance of genetic evidence in ASD, the outcomes that have been observed are listed below (Table 1):

Findings	**Author name**	**Year**	**Title of the study**
Functional disruption of a gene can affect tissues and can lead to high expression of genes that later on works as an identification of ASD candidate genes.	Ping wang	2018	Enriched expression of genes associated with autism spectrum disorders in human inhibitory neurons.
lncRNAs play crucial roles in ASD as their co-expression with ASD risk genes assist in identifying the candidate genes for ASD.	Steven B. Cogill	2018	Co-expression of long non-coding RNAs and autism risk genes in the developing human brains.

Involvement of X-linkage imprinting and sex hormones in ASD.	Donna M. Werling	2016	The role of sex differential biology in risk for autism spectrum disorder.
Male child being prone to predisposal of ASD by the increasing level of sex hormones and cortisol during fetal development.	Amanda Crider	2017	Estrogen signaling as a therapeutic target in neurodevelopmental disorders.
The opposite regulation of RORA receptor by males and protective effect of female hormones shows males being more prone to ASD	Tewarit Sarachana	2011	Sex hormones in ASD: Androgens and Estrogens differentially and reciprocally regulate RORA, a novel candidate gene for autism.

Table 1: List of findings from the review work

ASD candidate genes show enhanced expressions in neurons, particularly inhibitory neurons, during the developmental stage, thanks to the integration of ASD candidates and dysregulated genes in ASD samples, as well as down streaming targets. Inadequate function in these neurons can have a significant impact on function and contribute to the autism spectrum. The relative reduction in GABergic signaling reported in patients with ASD can be explained by an imbalance in the ratio of excitatory and inhibitory signaling. The elevated expression of ASD candidates in inhibitory neurons is inconsistent with the enriched expression of ASD samples, as their mutations result in reduced expression and functional loss. A possible reason appears to be the abnormal expression of ASD candidate that lead the inhibitory neurons in some specific region of brain to perform a compensation mechanism for the reduction of GABA receptor in individual inhibitory neurons (Wang et al., 2018).

Secondly, with the recent exploration of neurodevelopmental sectors and their connection with autism convey that ASD might also include other developmental disorders as – Asperger's Syndrome, Rett Syndrome, uncharacterized pervasive disorder and Autistic disorder. This information ends up increasing the complexity of the identification of the actual of cause of the autism spectrum disease. The transition from genetic anomalies to phenotypic causation has been difficult due to a variety of causes, including the disorder's intricacy of genetic interplay. Interestingly, lncRNAs (Long non-coding RNAs) have played a significant role in overcoming the challenges of discovering ASD candidate genes. lncRNAs play an important role in transcriptional control and synapse formation in the developing brain, as well as in ASD. Although lncRNAs were shown to be enriched across brain modules, they only co-enriched with ASD risk genes in two different module groups and have high prenatal and postnatal expressions, according to studies,

these expressions in particular period was proved to be critical time for ASD development and thus has marked lncRNAs as an excellent -biomarker for ASD (Cogill et al., 2018).

Furthermore, sex differences in the demonstration and occurrence of ASD symptoms are also consistent with the idea defining ASD risk factors having qualitative and quantitative difference in the impact with both males and females. Female specific or female -preferential protection is signified for reducing prevalence in ASD compared to males. The female protective mechanism is responsible for modulating the effects of risk factors and the difference in phenotype itself works profoundly in the protection. Other studies also show that ASD risk factors (gene mutation, fragile X syndrome, metabolic imbalance, gender, family history etc.) increase the risk in male only leading towards sex-differential prevalence. ASD is proved to be associated with the monogenic syndromes caused by X chromosomal mutations as- fragile X syndrome that affects mostly males because of having only one X chromosome in autosomal pair. The disruptive variations in the 65 genes discovered and their association with ASD risk demonstrate the distribution of disruptive variants among male and female probands, underpinning the genetic risk for ASD in males and females. Females are safeguarded by their second X chromosome, which has extra copies of the mutant risk genes that are likely functioning. Males do not express these genes because they lack the paternal X chromosome, hence they do not have the same protective features as females. Again, studies demonstrate a significant positive link between prenatal testosterone levels and ASD features, demonstrating the steroid hormone's role in ASD. Certainly, testosterone exposure causes a shift in trait distribution, which aids in the identification of diagnosable abnormalities. A significant factor is the abrupt increase in testosterone levels. The sudden elevation of testosterone is a key finding in affected male that separates them from unaffected males (Werling, 2016).

Studies on the impact of sex hormones on ASD shows increased level of sex hormones and cortisol during fetal development predispose male children to autism. The increased testosterone during pregnancy is responsible for the reduction of the estrogen receptor expression and this is expected to be correlated with ASD. Estradiol, which is created locally by testosterone conversion, is largely responsible for sex differences in the brain. Increased sensitivity to cognitive deficits occurs when estrogen signaling is reduced. This highlights the importance of brain-produced estrogen in neuronal activity. As a result of the decline, testosterone conversion to estradiol is hindered, resulting in elevated testosterone levels, as seen in ASD (Crider & Pillai, 2017).

Lastly, the study with RORA (RAR related orphan receptor A) receptor shows the aftermath of the difference in their regulation in respect of males and females. This is related to the increasing level of estradiol binding with AR (androgen) and ER (estrogen) to the RORA promoter region. Estradiol enhances RORA expression, whereas DHT (dihydrotestosterone) acts in repressing the expression. This remarkable mechanism and environment through which AR and ER regulate RORA in opposite directions is considered to be crucial in ASD diagnosis. Males are more sensitive to RORA deficiency due to the interplay between sex hormones and the RORA receptor. Aromatase, a catalyst enzyme for the production of estrogen from testosterone, is another transcriptional target of RORA. RORA deficiency is widespread in autism, and individuals are found to have a negative feedback system including a lowered aromatase level, which causes the accumulation of its substrate (testosterone) and the lowering of its product (estradiol). Finally, the findings confirm RORA's role as a candidate gene in ASD. Through aromatase control, it is involved in balancing male and female hormones in brain regions, and malfunction of this particular receptor is shown in ASD. On DNA and other common target genes, RORA and ER have a similar binding site. These findings demonstrate that estrogen not only protects females

from autism by boosting RORA expression, but also compensates for RORA deficiency (Sarachana et al., 2011).

These studies sum up the findings of co-expressions of specific brain regions, shows the effects of candidate genes with noteworthiness of gender specific genes and implicates the males being prone to ASD more than females. All the findings signify the genetic evidence of sex difference in ASD.

Chapter 4:

Conclusion:

Lastly, it can be said that there are various genetic evidence of sex difference regarding ASD. It includes the brain specific region, lncRNAs candidate genes, sex hormones, specific regions etc.

ASD being a neurological and developmental disorder, to underline the pathophysiology it is mandatory to study the genetic alterations and the relationship between genes. One of the goals of genetic research in ASD is to figure out what brain circuits cause phenotypic heterogeneity (Hashem et al., 2020). Then, a study of the lncRNAs candidate gene reveals the molecular processes that link several of the known autism candidate genes. These aid in delivering broadest efficacy in autistic patients by finding regulatory checkpoints and interconnecting transcriptional networks (Lanz et al., 2013). Again, a comprehensive investigation of estrogen and associated ligand expression in fetuses, particularly comparing sex, phenotype, and specific brain region, is critical in understanding the role of estrogen mediated transcription in sex-specific brain diseases such as ASD (Arnold & Saijo, 2021). Finally, the RORA receptor involvement in ASD shows the significance of sex difference as it is regulated by testosterone and estrogen respectively (Devanna & Vernes, 2014). These enlighten the aim of our study by showing the genetic evidence of sex difference in ASD. Moreover, by utilizing the genetic evidence, further steps can be taken regarding diagnosis and treatment of ASD. However, more research is needed to identify the unknown and uncertain areas of the findings to determine the actual use of genetic evidence in ASD.

To conclude, it is showed in the review that genetic evidence of sex difference plays crucial roles in ASD and it can help in understanding the pathology, diagnosis, probable cause and further treatment of ASD.

Limitations of the study:

- Some sources were unreachable as they were paid articles.
- Some of the studies aren't most recent ones.
- Expected number of articles couldn't be found by cross-referencing.
- Information regarding the topic were not available in context of Bangladesh.

Future study plan:

More study and investment in this field can be fruitful for future inventions and hopefully can solve the mystery behind neurological disease as ASD. In near future I want to discover more information in this field and research for underlying the uncertain sectors of this disease. If possible, I want to apply animal model to have better forecast of the research.

Reference:

Akhter, S., Hussain, A. H. M. E., Shefa, J., Kundu, G. K., Rahman, F., & Biswas, A. (2018). Prevalence of Autism Spectrum Disorder (ASD) among the children aged 18-36 months in a rural community of Bangladesh: A cross sectional study [version 1; referees: 1 approved, 2 approved with reservations]. *F1000Research*, *7*(0), 1–14. https://doi.org/10.12688/f1000research.13563.1

Al-Beltagi, M. (2021). Autism medical comorbidities. *World Journal of Clinical Pediatrics*, *10*(3), 15–28. https://doi.org/10.5409/wjcp.v10.i3.15

Ansel, A., Rosenzweig, J. P., Zisman, P. D., Melamed, M., & Gesundheit, B. (2017). Variation in gene expression in autism spectrum disorders: An extensive review of transcriptomic studies. *Frontiers in Neuroscience*, *10*(JAN). https://doi.org/10.3389/fnins.2016.00601

Arnold, M. L., & Saijo, K. (2021). Estrogen Receptor β as a Candidate Regulator of Sex Differences in the Maternal Immune Activation Model of ASD. *Frontiers in Molecular Neuroscience*, *14*(August), 1–8. https://doi.org/10.3389/fnmol.2021.717411

Cogill, S. B., Srivastava, A. K., Yang, M. Q., & Wang, L. (2018). Co-expression of long non-coding RNAs and autism risk genes in the developing human brain. *BMC Systems Biology*, *12*(Suppl 7). https://doi.org/10.1186/s12918-018-0639-x

Crider, A., & Pillai, A. (2017). Estrogen signaling as a therapeutic target in neurodevelopmental disorders. *Journal of Pharmacology and Experimental Therapeutics*, *360*(1), 48–58. https://doi.org/10.1124/jpet.116.237412

Devanna, P., & Vernes, S. C. (2014). A direct molecular link between the autism candidate gene RORa and the schizophrenia candidate MIR137. *Scientific Reports*, *4*(5), 1–7. https://doi.org/10.1038/srep03994

Hashem, S., Nisar, S., Bhat, A. A., Yadav, S. K., Azeem, M. W., Bagga, P., Fakhro, K., Reddy, R., Frenneaux, M. P., & Haris, M. (2020). Genetics of structural and functional brain changes in autism spectrum disorder. *Translational Psychiatry*, *10*(1). https://doi.org/10.1038/s41398-020-00921-3

Lanz, T. A., Guilmette, E., Gosink, M. M., Fischer, J. E., Fitzgerald, L. W., Stephenson, D. T., & Pletcher, M. T. (2013). Transcriptomic analysis of genetically defined autism candidate genes reveals common mechanisms of action. *Molecular Autism*, *4*(1). https://doi.org/10.1186/2040-2392-4-45

National Center on Birth Defects and Developmental Disabilities, C. for D. C. and P. (2021). *Signs @ Www.Cdc.Gov*. http://www.cdc.gov/healthyschools/noise/signs.htm

Rylaarsdam, L., & Guemez-Gamboa, A. (2019). Genetic Causes and Modifiers of Autism Spectrum Disorder. *Frontiers in Cellular Neuroscience*, *13*(August), 1–15. https://doi.org/10.3389/fncel.2019.00385

Sarachana, T., Xu, M., Wu, R. C., & Hu, V. W. (2011). Sex hormones in autism: Androgens and estrogens differentially and reciprocally regulate RORA, a novel candidate gene for autism. *PLoS ONE*, *6*(2). https://doi.org/10.1371/journal.pone.0017116

Verghese. (2011). 基因的改变NIH Public Access. *Bone*, *23*(1), 1–7. https://doi.org/10.1097/WCO.0b013e32835ee548.Sex

Wang, P., Zhao, D., Lachman, H. M., & Zheng, D. (2018). Enriched expression of genes associated with autism spectrum disorders in human inhibitory neurons. *Translational Psychiatry*, *8*(1). https://doi.org/10.1038/s41398-017-0058-6

Werling, D. M. (2016). The role of sex-differential biology in risk for autism spectrum disorder. *Biology of Sex Differences*, *7*(1), 1–18. https://doi.org/10.1186/s13293-016-0112-8

Williams, B. F., & Williams, R. L. (2019). Characteristics of Autism Spectrum Disorder. *Effective Programs for Treating Autism Spectrum Disorder*, 2–18. https://doi.org/10.4324/9780203855034-1

Zhang, Y., Li, N., Li, C., Zhang, Z., Teng, H., Wang, Y., Zhao, T., Shi, L., Zhang, K., Xia, K., Li, J., & Sun, Z. (2020). Genetic evidence of gender difference in autism spectrum disorder supports the female-protective effect. *Translational Psychiatry*, *10*(1). https://doi.org/10.1038/s41398-020-0699-8

Sumaia_Autism

ORIGINALITY REPORT

14%

SIMILARITY INDEX

PRIMARY SOURCES

4%

INTERNET SOURCES

10%

PUBLICATIONS

6%

STUDENT PAPERS

2%

Tewarit Sarachana. "Sex Hormones in Autism: Androgens and Estrogens Differentially and Reciprocally Regulate RORA, a Novel Candidate Gene for Autism", PLoS ONE, 02/16/2011
Publication

2%

Submitted to King's College
Student Paper

2%

Ping Wang, Dejian Zhao, Herbert M. Lachman, Deyou Zheng. "Enriched expression of genes associated with autism spectrum disorders in human inhibitory neurons", Cold Spring Harbor Laboratory, 2017
Publication

1%

Submitted to BRAC University
Student Paper

1%

Paul El-Fishawy, Matthew W. State. "The Genetics of Autism: Key Issues, Recent Findings, and Clinical Implications", Psychiatric Clinics of North America, 2010
Publication

orca.cf.ac.uk

1% Internet Source

1%

www.nature.com
Internet Source

1%

Duc-Hau Le, Nguyen Thi Van. "Meta-analysis of whole-transcriptome data for prediction of novel genes associated with autism spectrum disorder", Proceedings of the 8th International Conference on Computational Systems-Biology and Bioinformatics, 2017
Publication

1%

Submitted to newcastle under lyme college
Student Paper

<1%

Hemi Malkki. "Elevated fetal sex steroids might confer risk for autism", Nature Reviews Neurology, 2014
Publication

<1%

Submitted to Hong Kong Baptist University
Student Paper

<1%

Yi Zhang, Na Li, Chao Li, Ze Zhang et al. "Genetic evidence of gender difference in autism spectrum disorder supports the female-protective effect", Translational Psychiatry, 2020
Publication

<1%

Sheema Hashem, Sabah Nisar, Ajaz A. Bhat,
Santosh Kumar Yadav et al. "Genetics of structural and functional brain changes in autism spectrum disorder", Translational Psychiatry, 2020
Publication

Exclude quotes On Exclude bibliography On

Exclude matches < 4 words