AARHUS UNIVERSITY

Press release

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Basic information

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Department of: Clinical Medicine

Main supervisor: Professor Jens Randel Nyengaard

Title of dissertation: Investigation of Prefrontal Cortex Pathology in Df(h22q11)/+ and Df(h15q13)/+ Genetic Mouse Models of Schizophrenia

Date for defence: 04.02.2021 at (time of day): 14:00-16:00 Place: Online via Zoom

Press release (Danish)

Undersøgelse af præfrontal cortex patologi i Df (h22q11)/+ og Df (h15q13)/+ genetiske musemodeller af skizofreni

Skizofreni er en alvorlig lidelse med en enorm social og økonomisk byrde. Størstedelen af de aktuelt anvendte behandlinger mod skizofreni er kun delvist effektive. I løbet af de sidste år er kopiantalvariationer (CNV'er) blevet identificeret i det menneskelige genom, der disponerer til hjernesygdomme, herunder skizofreni. Blandt disse CNV'er findes både 22q11.2- og 15q13.3-deletioner, der begge øger risikoen for skizofreni signifikant. Hjernens præfrontal cortex (PFC) er en nøglestruktur, der regulerer flere af de funktioner, der er hæmmet i 22q11.2 og 15q13.3 deletions syndromer. Der er blevet lavet flere musemodeller, der afspejler de menneskelige 22q11.2 og 15q13.3 hemizygote CNVer, herunder Df (h22q11) / + og Df (h15q13) / + musemodellerne. Begge modeller viser flere funktionelle og adfærdsmæssige abnormiteter med relevans for skizofreni. Begge modeller karakteriseres ved forandringer, der påvirker forskellige cellulære komponenter i PFC. Samlet afslører PFC i modellerne Df (h22q11) / + og Df (h15q13) / + ændringer på molekylære, funktionelle og strukturelle niveauer, der involverer både neuronale og ikke-neuronale kredsløb. Resultaterne fra de to modeller identificerer molekylære substrater, der potentielt kan forklare en del af den skizofreni-relaterede fænotype, der observeres i 22q11.2- og 15q13.3-deletionssyndromer.

Forsvaret af Ph.d.-projektet er offentligt og finder sted online den 04 februar kl. 14.00-16.00 via Zoom. Se venligst nedenstående kontaktoplysninger, hvis link til forsvar ønskes. Titlen på projektet er ”Investigation of Præfrontal Cortex Pathology in Df(h22q11)/+ and Df(h15q13)/+ Genetic Mouse Models of Schizophrenia”. Yderligere oplysninger: Ph.d.-studerende Abdel-Rahman Al-Absi, e-mail: abd.alabsi@clin.au.dk, tlf. 71515087.

Bedømmelsesudvalg:
Professor Boldizsar Czeh, Szentagothai Janos Research Center, Department of Laboratory Medicine, Medical School, University of Pecs, Hungary.
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Press release (English)

Investigation of Prefrontal Cortex Pathology in Df(h22q11)/+ and Df(h15q13)/+ Genetic Mouse Models of Schizophrenia

Schizophrenia is a severe and debilitating disorder with tremendous social and economic burden. The majority of the currently applied treatments against schizophrenia are only partially efficacious. During the past years, copy number variations (CNVs) have been identified in the human genome that associate with multiple brain disorders, including schizophrenia. Among these CNVs, both the 22q11.2 and 15q13.3 deletions are reported to significantly increase the risk of schizophrenia. The brains prefrontal cortex (PFC) is a key structure known to regulate multiple functions that are impaired in the 22q11.2 and 15q13.3 deletion syndromes. Several mouse models have been generated that mimic the human 22q11.2 and 15q13.3 hemizygous deletions. This includes the Df(h22q11)/+ and Df(h15q13)/+ mouse models. Both models display multiple functional and behavioral abnormalities with relevance to schizophrenia. Findings from both models uncovered alterations affecting different cellular components of PFC. Collectively, PFC in the Df(h22q11)/+ and Df(h15q13)/+ models reveal changes at the molecular, functional, and structural levels implicating both neuronal and non-neuronal circuits. Results emerged from the two models identify molecular substrates that could potentially explain part of the schizophrenia-related phenotype observed in the 22q11.2 and 15q13.3 deletion syndromes.

The defence is public and takes place on February 04, 2021, at 14.00-16.00 online on Zoom. The title of the project is Investigation of Prefrontal Cortex Pathology in Df(h22q11)/+ and Df(h15q13)/+ Genetic Mouse Models of Schizophrenia. For more information including online access to the defence, please contact PhD student Abdel-Rahman Al-Absi, email: abd.alabsi@clin.au.dk, Phone +45 71515087.

Assessment committee:
Professor Boldizsar Czeh, Szentagothai Janos Research Center, Department of Laboratory Medicine, Medical School, University of Pecs, Hungary.
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