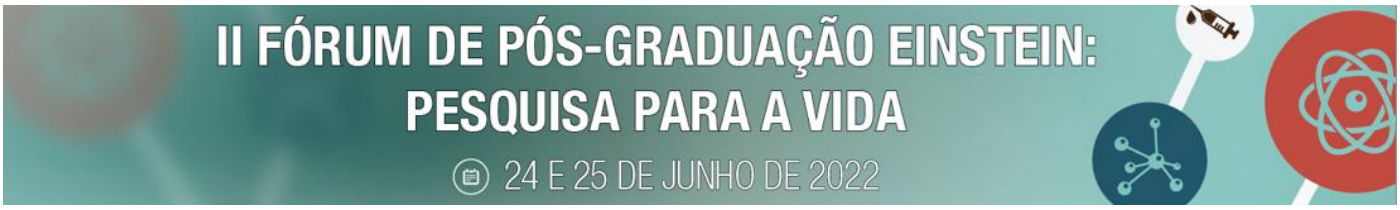

PÔSTERES VENCEDORES

Sete pôsteres foram indicados como melhores trabalhos, com nota máxima em todos os critérios.

A **Comissão Organizadora** estabeleceu como parâmetros para submissão dos resumos os Objetivos de Desenvolvimento Sustentável da ONU e a Agenda Nacional de Prioridades de Pesquisa em Saúde.

MELHORES PÔSTERES VENCEDORES

1.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 09. Indústria Inovação e Infraestrutura

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

09. Saúde dos portadores de necessidades especiais

ÁREA DE SUBMISSÃO DO PÔSTER:

Questões globais de saúde

Generation of urine-derived induced pluripotent stem cells and cerebral organoids for modeling Down syndrome

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Background: Down syndrome (DS), caused by chromosome 21 trisomy (T21), is the most common genetically defined cause of intellectual disability. The advent of human cerebral organoids (COs) grown from three-dimensional (3D) aggregates of induced pluripotent stem cells (iPSCs), that recapitulate many key aspects of human fetal brain development, has the potential to advance our understanding of the cellular and molecular mechanisms of DS. **Objective:** This study aims to generate urine-derived iPSCs from individuals with DS and euploid controls with the ability to differentiate into neurons and astrocytes in monolayer (2D) cultures as well as into 3D COs. **Methods:** Urine epithelial cells were reprogrammed with episomal vectors to generate iPSCs, according to Lee et al. (2017). Giemsa-banded karyotyping was performed in all iPSC lines. Differentiation of iPSCs into cortical neurons and astrocytes in 2D cultures, as well as into COs with dorsal forebrain identity was performed using established protocols (Shi et al., 2012; Hedegaard et al., 2020; Sloan et al., 2018). COs were collected on days 30, 60, 90, and 120 to perform cryosections for immunofluorescence analyses. Total protein from the COs were obtained for

western blotting. **Results:** We generated urine-derived iPSCs of three individuals with DS and three controls, and observed that all iPSC lines expressed key pluripotency markers, such as SOX2, LIN-28a, and OCT4. No chromosome abnormalities were observed in the control iPSC lines, and the presence of T21 was confirmed in the DS iPSC lines. We observed that both DS and control iPSC lines can be differentiated into 2D cultures of neurons positive for Synapsin-1 (SYN-1), microtubule-associated protein 2 (MAP2), and vesicular glutamate transporter-1 (VGLUL1), as well as 2D cultures of astrocytes positive for glutamine synthetase (Glut Synt), CD44, glial fibrillary acidic protein (GFAP), and the complement system component C4. Similarly to what occurs during *in vivo* early stages of cortical development, at day 30 we observed that DS and control COs exhibit well-defined ventricular zone (VZ)-like structures composed of SOX2-, Nestin-, and FOXG1-expressing neural progenitors cells. Also, these VZ-like structures were surrounded by MAP2-positive and SYN-1-negative immature neurons reminiscent of the preplate. At days 60 and 90, we observed that DS and control COs show a reduction of the VZ-like structures and formation of subventricular zone (SVZ)-like structures containing SOX2- and Nestin-positive neural progenitors, as well as MAP2- and SYN-1-positive neurons. At days 90 and 120, we detected the presence of both excitatory glutamatergic and inhibitory GABAergic neurons, which express VGLUT1 and gamma-aminobutyric acid type A receptor (GABAAR) respectively, as well as the presence of GFAP-, Glut Synt-, and C4-positive astrocytes in the COs from both groups. The temporal expression of key markers of neural progenitors, neurons, and astrocytes were confirmed by western blotting. **Conclusion:** Here, we show the successful generation of DS- and euploid-iPSC lines derived from urine cells, which can be harvested noninvasively, and their subsequent differentiation into 2D cultures of neurons and astrocytes, as well as into COs that model early developmental events in the human forebrain, and provide unprecedented opportunities for modeling DS.

2.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

02. Saúde mental

ÁREA DE SUBMISSÃO DO PÔSTER:

Questões globais de saúde

Rare CACNA1H and RELN variants interact through mTORC1 pathway in oligogenic autism spectrum disorder

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Background: Oligogenic inheritance of autism spectrum disorder (ASD) has been supported by several studies. However, little is known about how the risk variants interact and converge on causative neurobiological pathways. We identified in an ASD proband deleterious compound heterozygous missense variants in the Reelin (*RELN*) gene, and a de novo splicing variant in the Cav3.2 calcium channel (*CACNA1H*) gene. **Objective:** The main objectives of this work were to evaluate whether: 1) the variants in *RELN*and *CACNA1H*are functional and crosstalk through intracellular signaling pathways; 2) there is an increased burden of co-occurring risk variants in Reelin pathway and calcium channel genes in ASD. **Methods:** To evaluate the impact of the *CACNA1H*variant on the Cav3.2 channel structure, we built 3D models of the wild-type and mutant proteins. To assess the functional connectivity between the *RELN*and *CACNA1H*variants, we used iPSC-derived neural progenitor cells (NPCs) and a heterologous expression system in HEK293 cells, and we

analyzed gene and protein expression, extracellular calcium influx, cell morphology and size, cell proliferation and migration. Finally, to evaluate whether the concomitant occurrence of rare risk variants in Reelin pathway and calcium channel genes is enriched in ASD, we analyzed of the sequencing data from two ASD cohorts - a Brazilian cohort of 861 samples, 291 with ASD; the MSSNG cohort of 11,181 samples, 5,102 with ASD. **Results:** We show that the variant in Cav3.2 leads to significant structural changes in the mutant channel, resulting in larger open pores, which causes increased calcium influx into cells, overactivates mTORC1 pathway and, consequently, further exacerbates the impairment of Reelin signaling. Also, we show that Cav3.2/mTORC1 overactivation induces proliferation of NPCs and that both mutant Cav3.2 and Reelin cause abnormal migration of these cells. Finally, analysis of the sequencing data from the Brazilian and MSSNG cohorts revealed that the co-occurrence of risk variants in both alleles of Reelin pathway genes and in one allele of calcium channel genes confer significant liability for ASD. **Conclusion:** Our results support the notion that genes with co-occurring deleterious variants tend to have interconnected pathways underlying oligogenic forms of ASD.

3.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

05. Doenças não-transmissíveis

ÁREA DE SUBMISSÃO DO PÔSTER:

Questões globais de saúde

The imbalance in systemic CD4 T cell frequency and granzyme B expression can predict immune-related adverse events in NSCLC patients treated with immune checkpoints immunotherapy

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Immune checkpoint inhibitors (ICI) immunotherapy has been in the oncology treatments' spotlights due to its benefits in patients with some solid advanced/refractory tumors when compared with traditional therapies. Although immunotherapy brings new opportunities for oncologic patients, the treatment course can be marked by a systemic extra tumoral inflammation known as immune-related adverse events (irAEs). This outcome besides being life-threatening can impact directly the therapeutic approach by interruption or administration of corticosteroids which reduces the anti-tumoral response. Currently, metastatic non-small-cell lung cancer (NSCLC) treated with pembrolizumab (anti-PD-1) plus chemotherapy (carboplatin/cisplatin + pemetrexed) as first line therapy and shows approximately 48% of response rate and about 10% of severe irAEs. Even though most studies focused on understanding response mechanisms, the causes of irAEs remain unknown, especially in NSCLC. **OBJECTIVE:** Identify possible biomarkers and immune mechanisms involved in irAEs development in NSCLC patients treated with ICI. **MATERIALS AND METHODS**: 25 patients diagnosed with NSCLC candidates for first line immunotherapy and treated at A.C Camargo

Cancer Center were included according to institutional ethical approval #2671/19. Patients were graded during treatment by an oncologist according to the toxicity degree (Grades 0 to 5) and grouped into No/Mild (G0- G2, n=20) and Severe (G3-G4, n=5). Using peripheral blood samples collected at baseline, we characterized peripheral blood mononuclear cells by multiparametric flow cytometry (FACSymphony A5, BD Bioscience) after polyclonal stimulation. We compared the immune cell populations frequencies between No/Mild and Severe using appropriate tests and considered statistical difference when p-valueRESULTS: The median of irAEs time onset was 32.7 weeks (3 – 69.9). Aiming to identify potential differential activation/suppressive markers between No/Mild and Severe we found that patients with Severe toxicity had higher expression of granzyme B in viable leukocytes. We also performed manual gating that returned an increased frequency of CD4 T cells in No/Mild patients (Median: 38.75%; 95% CI: 30.60 – 45.60, against 25.10%; 17.10 – 36.60 in Severe) compared to Severe. In contrast, the Severe group showed a higher frequency of granzyme B+ cells in TCRαβ T cells, NK and CD8 effector T cells (76.90%; 61.90 – 93.80) compared to the No/Mild group (58.65%; 36.50 – 64.90). Based on these results, we evaluated the capacity of these populations to predict irAEs development using simple and multiple logistic regression. The combination of CD4 T cells and CD8 effector T cells GzB+ showed a good model of prediction with a ROC curve's AUC of 0.88, odds ratio of 0.87 (95% CI: 0.70 - 0.99) and 1.09 (95% CI: 1.01 - 1.24), respectively, negative predictive power of 90.48% and positive predictive power of 75%. **CONCLUSION**: Our results highlighted the imbalance between immune populations in irAEs development, suggesting a cytotoxic profile in Severe patients and a possible immunoregulatory profile in No/Mild before treatment initiation. Both characteristics, in the future, may be used together as good parameters to predict irAEs.

4.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

02. Saúde mental; 09. Saúde dos portadores de necessidades especiais

ÁREA DE SUBMISSÃO DO PÔSTER:

Questões globais de saúde

The role of complement component C4 on human astrocyte functions

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Background: Accumulating evidence has shown that the innate immune complement system is involved in several aspects of normal brain development and in neurodevelopmental disorders, including autism spectrum disorder (ASD). Recently, we have found that complement-component C4 mRNA and protein are expressed in significantly lower levels by induced pluripotent stem cell (iPSC)-derived astrocytes of individuals with ASD. As astrocytes participate in synapse elimination and diminished C4 levels have been linked to defective synaptic pruning, it is possible that reduced C4 expression by ASD-derived astrocytes may contribute to the atypically enhanced brain connectivity in ASD. However, the impact of reduced C4 expression on human astrocyte functions is still largely unknown. In humans, C4 is encoded by two different genes, *C4A* and *C4B* , which are located in tandem on chromosome 6 and vary in copy number. **Objective:** The aims of this work are to generate knockout alleles of the *C4A/B*genes in human iPSCs by using the CRISPR-Cas9 technology; and to verify whether iPSC-derived astrocytes with reduced expression of C4 show abnormalities in differentiation, morphology, proliferation, and/or secretion of pro-inflammatory cytokines. **Methods:**We analyzed the expression of C4, as well as of the astrocyte marker GFAP, in iPSC-derived astrocytes of control individuals by immunocytochemistry. We are using CRISPR-Cas9[2] to knockout copies of the *C4A/B*genes in human control iPSC lines. The

CRISPOR designer tool was used to design the guide RNAs (gRNAs). The plasmid vector pSp-Cas9(BB)-2A-Puro (PX459) was used to clone the gRNAs for expression of a chimeric gRNA plus a puromycin-resistance marker and human Cas9. Two different protocols were used for cloning the gRNAs, the Nature's protocol, and a digestion–ligation protocol, which uses a greater number of steps. Competent bacterial cells were transformed with the PX459 plasmids containing the gRNAs, and the integrity of the constructs were verified by Sanger sequencing. To select the gRNAs with the highest editing rates, the constructs were transfected in HEK293T cells, and genomic DNA was extracted for TIDE assay. **Results:** iPSC-derived astrocytes from healthy donors have been previously generated. Here, we observed by immunocytochemistry the expression of C4 and GFAP in these iPSC derived astrocytes. Using CRISPOR, we selected four gRNAs that target both *C4A*and *C4B*, and two gRNAs specific to *C4B*. These gRNAs were then inserted into the PX459 vector, and we observed that 5 out of 6 gRNAs were cloned successfully using the two different cloning protocols. HEK293T cells were transfected with the plasmids containing gRNAs, and genomic DNA was collected for determination of Cas9 cutting efficiency (TIDE assay). Once the gRNAs with the highest editing rates are selected, they will be used for transfection of iPSCs, and then the *C4A/B*-edited iPSC lines will be used to generate astrocytes for functional analysis. **Conclusion:** We observed expression of C4 in human iPSC-derived astrocytes by immunocytochemistry, which has not been shown previously. We also designed and cloned gRNAs targeting the human *C4A/B*genes. The results obtained here will provide a better understanding of the consequences of decreased astrocyte-derived C4 for brain development and ASD pathophysiology.

5.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

23. Saúde, ambiente, trabalho e biossegurança

ÁREA DE SUBMISSÃO DO PÔSTER:

Inovação em Saúde

GLIOBLASTOMA ON-A-CHIP FOR THERAPEUTIC APPLICATIONS

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Glioblastoma is one of the most malignant types of central nervous system tumors. Despite therapeutic advances, the prognosis is threatening, with a median survival time of about 10-14 months after diagnosis. Objective: To develop a microfluidic device capable of recreating the structural and physiological organization of glioblastoma tissue and stroma for the evaluation of therapeutic applications in Glioblastoma on-a-chip. Methods: The development of the microfluidic device involved four steps: in the first step, the organ- on-a-chip design was created, in a python programming language by script in the CleWin software, in order to make changes in the device dimensions automatically. In the second step, the fabrication of microfluidic channels in PDMS (Polydimethylsiloxane) was carried out from the channel geometry designed in CleWin® software and transferred into a SU-8 master mold on silicon by photolithography in MicroWriter®. Then, the geometry was validated using the Dektak XT® perfilometer and micromolding of the PDMS (Sylgard 184®) was performed. After the PDMS was demolded and the entry/exit holes were opened, they will be used for the entry of cells, extracellular matrix, and the culture medium. In the third step, the sealing process of the device was performed by plasma treatment under N_2O atmosphere, juncting the device in PDMS and a glass slide. Finally, the 3D

co-culture of cells was performed, at this stage with the microfluidic device finished, the U87 MG cells and endothelial cells were co-cultured in DMEM-F12 supplemented with geltrex and fibronectin, in the central channel in a 2:1 ratio and so on for 14 days in culture the glioblastoma tumor was developed in the microfluidic device. Results: The microdevice developed presents a channel with a circular chamber in the central region and two lateral channels positioned in parallel. In these channels, a functional communication between the central chamber and the lateral channels through pores was demonstrated. Geometry validation showed that the channels are in the margin of error in the design. After validation, the cells were infused through the device entrance (epithelial and tumor cells in the central channel) together with the extracellular matrix and the medium exchange was performed through the lateral (external) channels for a continuous period of 12 h with interruption of the flow in 4 h periodically, for a total time of 14 days. The microscopy images demonstrated the structural organization of the cells, evidencing the need to standardize the flow rate of the infused medium in the lateral channels (µl/min), so that the exchange of substances occurs, so that the pressure exerted on the cells is unable to remove them from their adhesion to the matrix, so in this way, we have the glioblastoma on-a-chip implemented for its further use in therapeutic applications. Conclusion: The Glioblastoma on-a-chip microfluidic device was developed adequately, with a central part for the glioblastoma and tumor environment formed and at the border of the central part a pores and channel system that can simulate transport through the blood-brain barrier and thus be able to test different therapies in on glioblastoma on-a-chip.

6.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

05. Doenças não-transmissíveis

ÁREA DE SUBMISSÃO DO PÔSTER:

Inovação em Saúde

Identification of plasmatic biomarkers with prognosis and diagnosis potential for meningioma

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Objective: Given the absence of assertive and accessible diagnostic methods capable of identifying meningiomas in its early stages of development, as well as minimally invasive techniques for grade identification, the study aimed to detect and identify differential biomarkers related to metabolic alterations due to the process of carcinogenesis and evolution to malignant tumors through metabolomics analysis of the patient's plasma.
Methods: Blood samples were collected from 51 meningioma patients and 50 healthy donors, and the plasma was collected. Plasmatic metabolites were extracted and ionized for direct injection into mass spectrometer for analysis in positive mode, in the mass range of 100 and 1400 *m/z*(mass/charge). The selected molecules were submitted to fragmentatio for structural verification and subsequent identification by searching metabolites on databases and scientific literature. Spectral data were submitted to *in silico*analysis using the MetaboAnalyst platform and the PLS-DA was applied to verify the most important biomarkers for MGM group (diagnostic biomarkers), what was achieved through Variable

Importance in Projection (VIP) score, as well as differential biomarkers among the different grades of meningioma (prognostic biomarkers). Univariate Fold Change (FC) and Test-t analysis were also performed for the selected markers. Intending to verify markers' diagnostic and prognostic potential, the ROC curve was built for both groups.

Results: According to the VIP score, the six most important metabolites were selected as potential diagnostic biomarkers and we selected those with p-value < 0.05 and FC >2.5, resulting in four candidates: *m/z*143, 1116, 102, and 931. Therefore, the ROC curve was built for these molecules, generating an Area Under the Curve (AUC) of 0.997 (99.7%). About prognostic biomarkers, those with p-value < 0.05 and FC >1.5 were selected, resulting in five molecules with enhanced intensity in high grade meningioma patients (*m/z*294, 908, 800, 910, and 985). The ROC curve was built for prognostic markers, generating an AUC of 0.957 (95.7%). Evaluating the biological role of the selected molecules, a ganglioside (*m/z* 1116) and a sulfatide (*m/z* 931) were among the most relevant molecules for the diagnosis of meningioma in the present work. Respectively, a lipid from the neural cell membrane and a by product of ceramide metabolization might represent structural and biochemical modifications during cancer development, specially sulfatide, which indirectly represents a failure in tumor suppression usually induced by ceramides. Regarding to prognostic biomarkers, besides sulfatide (*m/z* 908), we identified lactosylceramide (m/z *985*;) and phosphatidylserines (*m/z* 800 and 910). Lactosylceramide and sulfatide, both byproducts of ceramide metabolism, could indicate a suppressed antitumor response in high grade meningioma patients. Also, phosphatidylserine is involved in anti-inflammatory responses through its externalization at the lipid membrane, also a possible signal of inhibited immunological response in malignant tumors.

Conclusion: Given the importance of early diagnosis and grade detection of CNS tumors for meningioma prognosis, the present study was able to select and identify potential diagnostic and prognostic biomarkers. Taken together, our data could be used for the development of more assertive, accessible, minimally invasive, and early diagnostic and grade detection methods for meningioma.

II FÓRUM DE PÓS-GRADUAÇÃO EINSTEIN:
PESQUISA PARA A VIDA
24 E 25 DE JUNHO DE 2022

7.

OBJETIVOS DE DESENVOLVIMENTO SUSTENTÁVEL – ONU

Objetivo 03. Saúde e Bem Estar

AGENDA NACIONAL DE PRIORIDADES DE PESQUISA EM SAÚDE

05. Doenças não-transmissíveis

ÁREA DE SUBMISSÃO DO PÔSTER:

Outros

Molecular characterization of the antineoplastic effect of curcumin on acute myeloid leukemia cell lines

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Objective: To evaluate the antileukemic effect of curcumin on leukemic cell lines and identifygenes andpathwaysassociatedwithcytotoxicity.**Methods:** Theleukemiccelllines HL-60, THP-1, OCI-AML2 and OCI-AML3 were treated with curcumin at different concentrations and evaluated for cell death and cell cycle changes by means of flow cytometry based assays. Pre- and post-treatment samples were used for RNA extraction, transcriptome sequencing and subsequent WGCNA analyses. **Results:** Curcumin causes variable degrees of cell death (sub-G0 phase of the cell cycle) at various concentrations. Wholetranscriptomeanalysisrevealedthatcurcumintreatmentinducedanincreaseinthe expression of genes associated with the proteasome, cell cycle, TNF and NF-KB pathways. **Conclusion:** Curcumin causes cell death in acute myeloid leukemia cell lines and this cytotoxiceffectisassociated withtheoverexpressionofgenesassociatedwithproteasome, cellcycle,TNFandNF-KBpathways.These results may contribute to the generation of new hypotheses regarding the anti-leukemogenic action of curcumin.