HUMAN GENOME AND STEM CELL RESEARCH CENTER (HUG-CELL)

Universidade de São Paulo

Instituto de Biociências

Departamento de Genética e Biologia Evolutiva

FAPESP/CEPID 2013/08028-1

Coordinator: MayanaZatz

REPORT 5

July 2017 to December 2018

SUMMARY

GROUP MEMBERS

Coordination

MayanaZatz - General coordinator

Maria Rita Passos-Bueno - Transfer of Technology

Eliana M. BelluzzoDessen - Education/Public

Principal investigators since 2016

Carla Rosenberg

Esper Cavalheiro

Mariz Vainzof

Merari de Fátima Ramires Ferrari

Oswaldo Keith Okamoto

Regina Célia Mingroni Netto

New Principal investigators

Ana Krepischi

Debora R. Bertola

Associate Investigators since 2016

Andrea L. Sertie

Angela M. Vianna Morgante

Carlos Frederico Menck

Célia P. Koiffmann

Edson Amaro Jr.

Fernando Kok

Luciana Haddad

Luis Eduardo Netto

Maria Vibranovski

Miguel Mitne-Neto

Peter Lees Pearson

Yeda Duarte

Valdemir Carvalho

ABSTRACT

Since July 2017 our group published 97 articles in peer-review journals, 2 book chapters, 22 Abstracts in National meetings and 42 in international meetings. Our students submitted 7 MSc Dissertations and 5 PhD Theses . Most of the articles involved the collaboration of students and PIs from **HUG-CELL**. About 37 conferences, lectures and symposia were done by our time.

In addition to the ongoing projects, we have embarked in a project on zika virus infection and the role of the host genome in the development of microcephaly and/or other associated malformations, namely, zika congenital syndrome. This project involved several **HUG-CELL** members as well as other groups of researchers, and was published in Nature communication this year. We have also shown that zika virus can be a potent oncolytic agent against brain tumors, which can open new avenues for therapies. These results were published and were the cover of the journal Cancer Research (Kaid et al., June 2018). Our citation index per year continues to increase as observed in https://scholar.google.com.br/citations?hl=pt-BR&user=CH1QvYIAAAAJ&view_op=list_works&sortby=pubdate)

The applications of technology transfer included genetic counseling for about 1270 families. A total of 632 NGS tests and ~48,632 sanger sequencing/microsatellite reactions, were performed at HUG-CEL EMU for 9 researchers, ~250 external users and for genetic diagnosis from our non-profit laboratory. As part of our program to train a larger number of persons for NGS analysis and interpretation, an international course in collaboration with University of Leiden on this topic was offered for 90 students in November /2018 at HUG-CELL.

Our education program included several projects such as laboratory classes at public schools, the Giant Cell project, educational leaflets, TV programs, among others. Furthermore, the **Sowing the seed of knowledge Project,** which aims to disseminate science knowledge and curiosity in subways and other public spaces, which was started in HUG-CELL was extended to other CEPIDS, under the coordination of Eliana Dessen.

PART 1
RESEARCH

Our main research results from July 2017 to December 2018, ordered by our main objectives and revised according to the Advisory Committee's recommendations are presented below.

A. GENE IDENTIFICATION AND MECHANISMS IN GENETIC DISORDERS

A1. Identification of new human genes in both simple (Mendelian) and complex *disorders*

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A. GENE IDENTIFICATION AND MECHANISMS IN GENETIC DISORDERS

A1. Identification of new human genes in Mendelian and complex *disorders*

A1.1. *Mendelian Disorders*

PTEN and hereditary primary microcephaly (HPMC)

HPMC is mainly characterised by decreased occipitofrontal circumference and variable degree of intellectual disability. We identified a family with three members affected by autosomal dominant MCPH.

A 382 kb microduplication at 10q23.31 was detected, encompassing the entire *PTEN*, *KLLN* and *ATAD1* genes. *PTEN* haploinsufficiency has been causally associated with macrocephaly and autism spectrum disorder and, therefore, was considered the most likely candidate gene to be involved in this autosomal dominant form of MCPH. In patients' fibroblasts, *PTEN* mRNA and protein were found to be overexpressed, and the phosphorylation patterns of upstream and downstream components of the mammalian target of rapamycin (mTOR) signalling pathway were dysregulated.

Our results suggest that the most probable pathomechanism underlying the microcephaly phenotype in this family involvesdownregulation of the mTOR pathway through overexpression of *PTEN (Oliveira et al, J. Med. Genet, 2018)*.

KIF5A, a novel amyotrophic lateral sclerosis (ALS) Gene

A genome-wide association study comparing 20,806 ALS cases and 59,804 controls is currently underway by an International Consortium. Independently, we performed a rare variant burden analysis comparing 1,138 index familial ALS cases and 19,494 controls. Through both approaches, we identified kinesin family member 5A (KIF5A) as a novel gene associated with ALS. Interestingly, mutations predominantly in the N-terminal motor domain of KIF5A are causative for two neurodegenerative diseases: hereditary spastic paraplegia (SPG10) and Charcot-Marie-Tooth type 2 (CMT2). In contrast, ALS-associated mutations are primarily located at the C-terminal cargo-binding tail

domain and patients harboring loss-of-function mutations displayed an extended survival relative to typical ALS cases. Taken together, these results broaden the phenotype spectrum resulting from mutations in KIF5A and strengthen the role of cytoskeletal defects in the pathogenesis of ALS. This work, published in Neuron is the result of our participation in this international consortium *(Nicolas et al., Neuron, 2018)*

A new *UBE2A* mutation: demonstration of impaired function of the mutated protein and *in vitro* reversion of the defect

A novel pathogenic missense mutation (Q93E) in the E2-conjugating enzyme UBE2A was identified in two brothers presenting mild intellectual disability. In collaboration with LNBio (Brazilian Biosciences National Laboratory), it was found an impairment in aminolysis by the mutated protein but no effect on the ability of UBE2A to conjugate with ubiquitin. However, aminolysis activity of the Q93E mutant is observed at high pH, providing the first evidence of a potential reversion of a defective mutation in *UBE2A. (Oliveira et al, in press)*

Novel CAPN1 mutations in hereditary spastic paraplegia 76

Mutations in CAPN1 may lead to pure or complicated autosomal recessive (AR) hereditary spastic paraplegia (HSP), classified as spastic paraplegia 76 (SPG76, OMIM # 616907). In the past two years, several groups have identified SPG76 patients. We identified eight additional SPG76 patients and compared them with 24 recently reported cases. Our data support that SPG76 is characterized mainly by lower-limb spasticity, ataxia and dysarthria. Upper-limb spasticity was observed in half of the patients and should be examined in order to better characterize clinically candidate patients for SPG76. In short, we reinforce the importance of screening the CAPN1 gene using next-generation sequencing in individuals with AR-HSP. There results were published in Clinical Genetics *(Melo et al., 2018).*

Syndromic and non-syndromic hearing loss

Several contributions were made to the genetic characterization of patients with syndromic and non-syndromic hearing loss. Novel mutations were found and unusual cases and families were described (Nonose et al., 2018; Bocangel et al, 2018 and Soares de Lima et al., 2018). However, the most relevant recent contribution to the understanding of genetic hearing loss was the finding of a novel mutation in the *MYO3A* gene. *MYO3A* has been long known for its relation to autosomal recessive hearing loss, with only one exceptional description of dominant inheritance. We described, in two large Brazilian families, a novel mutation in *MYO3A* leading to hearing loss, with dominant transmission. Functional studies performed in collaboration with the NIH-NIDCD (National Institute on Deafness and Other Communication Disorders), USA, showed that the mutant protein has dominant negative effect over the wildtype protein and this explains the hearing loss phenotype in heterozygotes. The screening of the mutation identified three additional families with the same mutation, which allowed investigation of the origin and age of the mutation *(Bueno et al., 2018).*

Another relevant contribution of the period was the identification and description of a junctional protein network which includes the protein Connexin-26, the product of the gene that most frequently explains congenital hearing loss. We used the Connexin-26 protein as a bait to capture its partners by affinity capture, followed by their identification through mass spectrometry. Many detected interactions were confirmed by co-immunoprecipitation. The study allowed the conclusion that Connexin 26 associates with components of other membrane junctions that integrate with the cytoskeleton *(Batissoco et al .2018)*. The twelve Connexin 26 partners identified in the study are potentially involved in hearing and their corresponding genes are good novel candidates to be explored in cases of genetic hearing loss.

SANTOS syndrome with limb defects

In 2017, we identified a mutation in the *WNT7A* gene as the cause of Santos syndrome *(Alves et al., 2017)*, described in Brazil in 2008 (MIM

613005). The *WNT7A* gene had already been related to different syndromes with limb defects. Its characterization as the cause of Santos syndrome contributed to broaden our understanding about the phenotypic spectrum of syndromes with limb defects.

Skeletal Dysplasias

- FN1

In a sporadic case presenting an autosomal dominant rare skeletal disorder (spondylometaphyseal dysplasia with"corner fractures"), the trio exome sequencing revealed a de novo variant in gene (FN1) that codes for a protein (fibronectin) that is a component of the extracellular matrix. We collaborated with a group from the University of Montreal in Canada that was already performing functional analysis of the variants found in the same gene in individuals with this skeletal disorder, increasing to seven, the number of families found to harbor variants in FN1. This discovery showed that defective fibronectin secretion is the cause of this rare skeletal dysplasia *(Lee et al., 2017)* .

- PLOD2

In a collaborative study with a colleaguefrom the Northeast of Brazil and Interantional groups from Sweden and Japan we we able to expand the phenotypic spectrum of skeletal disorders associated with biallelic variants in PLOD2, leading to a wide variety of skeletal dysplasias with bone fractures *(Leal et al., 2018).*

A1.2. *Complex disorders*

Copy number alterations in congenital disorders

CNVs are known to contribute to human normal variation and disease. Genomic imbalances have been investigated in several cohorts to identify genes or chromosomal regions involved in:, autosomal dominant microcephaly *(Malvezzi et al, 2018; Oliveira et al, 2018)*, congenital hypopituitarism *(Correa et al, 2018)* and short stature *(Homma et al, 2018).*

We also improved the identification of copy number alterations from

target sequencing data. Our data show that the combination of a sequencing platform comprising focused exome and whole genome backbone, with appropriate algorithms, enables a cost-effective and efficient solution for the simultaneous detection of CNVs and SNVs *(Villela et al, 2017).*

Copy Number Variation Mosaicism in Elderly Human Brain

Previous reports have shown that the genome of neuronal cells displays somatic genomic mosaicism including DNA copy number variations (CNVs). In the present study, we demonstrate a highly significant increase in the number of CNVs in nondiseased elderly brains compared to the blood. In two neural tissues isolated from paired *postmortem* samples (same individuals), we found a significant increase in the frequency of deletions in both brain areas, namely, the frontal cortex and cerebellum. Nearly all evidence of genome structural variation in human brains comes from studies detecting changes in single cells which were interpreted as derived from independent, isolated mutational events. The observations based on array-CGH analysis indicate the existence of an extensive clonal mosaicism of CNVs within and between the human brains revealing a different type of variation that had not been previously characterized *(Villela et al, 2018).*

Diabetes and Alzheimer's Disease Neuropathology

Previous evidence linking diabetes to Alzheimer's disease (AD) neuropathology is mixed, and scant data are available from low- and middle-income countries. Therefore, the aim of our study was to investigate the association between diabetes and AD neuropathology in a large autopsy study of older Brazilian adults.

Among 1,037 subjects , diabetes was present in 279 subjects. Diabetes was not associated with BB or with CERAD scores on analyses adjusted for sociodemographic and clinical variables. We observed effect modification by the APOE allele ε4 on the association between diabetes mellitus and BB scores.

In short, no evidence of an association between diabetes and AD neuropathology was found in a large sample of Brazilians; however, certain subgroups, such as APOE allele ε4 carriers, had higher odds of accumulation of neurofibrillary tangles. *(Dos Santos Matioli et al., J. Alzheimer Dis, 2017)*

Genetic alterations affecting cancer development and aggressiveness

While isolated genes can account for selection of specific chromosome imbalances (drivers), another alternative theory, applying an evolutionary perspective, hypothesizes that the different karyotypes with specific combinations of chromosome alterations could result in slightly different tumor subtypes, and progression. We investigated the role of copy number alterations in hepatoblastomas *(Aguiar et al, 2017*), myelodysplasia *(Silva et al, 2018)* and squamous cell carcinoma ex pleomorphic adenoma *(Mariano et al, 2018)* identifying genes and chromosome regions associated with tumor development and progression as well as clinical features.

Tumor development and aggressiveness may also involve aberrant expression of stemness genes. We have reported the establishment and characterization of a new cell line derived from a medulloblastoma patient with advantageous features for pre-clinical studies, namely enhanced aggressive traits, stem cell properties, increased chemoresistance, tumorigenicity in an orthotopic metastatic model, and resemblance of original medulloblastoma behavior *(Silva et al. 2016).* Employing this novel cell line, in addition to other classic cell lines available in central repositories, in functional studies, we found a specific correlation between OCT4A expression and poor survival, as well as a potent oncogenic activity for OCT4A, which enhanced metastatic spreading of tumor cells within the neural-axis. OCT4A expression also contributed to acquisition of heterogeneous chromosomal aberrations and aberrant expression of non-coding RNAs in stem-like cancer cells *(Silva et al. 2017).* These findings highlight the involvement of OCT4A in a mechanism driving aggressiveness of medulloblastoma, which could be further explored not only as a prognostic indicator, but also as a therapeutic target for a precision medicine approach in neuro-oncology.

Finally, we have also investigated genetic mechanisms involved in cancer resistance to genotoxic agents. The maintenance of genome stability is highly relevant to prevent cancer, but tumor cells use these mechanisms to resist to genotoxic anti-tumor chemotherapeutic drugs, such as cisplatine and temozolomide (TMZ). A role of the NADPH oxidase DUOX1 in the carcinogenesis process in breast cells, including increase in cancer features, was revealed by shRNA silencing of *DUOX1* expression. *(Fortunato et al, 2018)*

Rare variants in craniofacial complex disorders

Non-syndromic cleft lip and palate (NSCLP) is a common complex disorder (1: 600 births) with still unsolved genetic architecture. Using a novel approach, expression quantitative trait analysis and case-control study, we identified a new variant in a novel candidate locus (MRPL53) that contributes to the etiology of NSCLP. This variant is of Ameridian ancestry *(Masotti et al., 2017).*

Mosaiscim, new pathogenic variants and altered cytoskeleton organization in Autism spectrum disorder (ASD): ASD is a genetic heterogenous complex disorder and in the last years genomics had greatly contributed to the understanding of the genetic architecture of ASD. It has also been shown the need to establish large consortiums in order to get conclusive data. In this regard, our group has recently been invited to participate in the Autism Sequencing Consortium and our data has contributed to the study of mosaicism in ASD *(Lim et al., 2017)*. In addition, our group, in collaboration with two CEPID associated researchers (Dr. Sertie and Dr. Griesi-Oliveira) had recently demonstrated the pathogenicity of two novel mutations in RELN based on cell functional analysis; in this same study, we also showed which are the main dysregulated pathways, and provided evidence of the oligogenic model in ASD *(Sanchez-Sanchez et al., 2018*). In addition, in cells of ASD individuals, we demonstrated dysregulation of the cystoskeleton, which is critical for appropriate axonal differentiation, which in turn, is critical for a normal functional brain *(Griesi-Oliveira et al., 2018).* Both studies were based on stem cells from patients, as compared to control cells (iPSC-derived neuronal cells and

mesenchymal stem cells).

Congenital Zika syndrome susceptibility: what did we learn from twin studies?

Congenital Zika syndrome (CZS), caused by Zika virus (ZIKV) infection, has been associated to impairment of early brain development, particularly related to neural progenitor cells (NPC) survival and growth. However, no study has reported the outcome of NPC derived from CZS affected and non-affected babies exposed to ZIKV. Reports on discordant dizygotic twins (DZ) whose mothers were infected by ZIKV during pregnancy suggest that host genomic variants may contribute to the development of CZS. We investigated eight pairs of twins born from mothers with gestational ZIKV infection: five are discordant (affected and non-affected), all dizygotic (DZ) while three are concordant (both affected), one DZ and two monozygotic (MZ). Human induced pluripotent stem cells (hiPSC)-derived NPC from three pairs of dizygotic/discordant twins for CZS were infected with Brazilian ZIKV ($ZIKV^{BR}$). We demonstrated for the first time that NPCs from the affected twins had significantly higher viral release, reduction of cell proliferation and impaired mTOR signaling, as compared to their respective non-affected siblings. Through whole-exome sequencing analysis no rare variant of moderate-large effect was identified, suggesting the multifactorial inheritance is the most likely genetic mechanism to explain the genetic susceptibility to CZS upon maternal infection during pregnancy *(Caires-Junior et al., Nature communication 2018).*

This work involved many students from our CEPID and several groups of investigators from different Brazilian states. It received an award from ISSCR and a great attention from local and international media

A2. Elucidation of mechanisms to explain phenotype, clinical variability, and non-penetrance in genetic disorders

A2.1. Neuromuscular disorders

A novel complex neurological phenotype due to a homozygous mutation in FDX2.

Mutations in a number of genes that encode proteins involved in mitochondrial [Fe-S] protein assembly lead to complex neurological phenotypes. One class of proteins essential in the early cluster assembly are ferredoxins. FDX2 is ubiquitously expressed and is essential in the de novo formation of [2Fe-2S] clusters in humans. We identified six patients from two apparently unrelated families with autosomal recessive inheritance of a complex neurological phenotype involving optic atrophy and nystagmus developing by age 3, followed by myopathy and recurrent episodes of cramps, myalgia and muscle weakness in the first or second decade of life. Sensory-motor axonal neuropathy led to progressive distal weakness. MRI disclosed a reversible or partially reversible leukoencephalopathy. Muscle biopsy demonstrated an unusual pattern of regional succinate dehydrogenase and cytochrome c oxidase deficiency with iron accumulation. In both families, the phenotype was mapped to the same homozygous missense mutation in FDX2 (c.431C > T, p.P144L). The deleterious effect of the mutation was validated by real-time reverse transcription polymerase chain reaction and Western blot analysis, which demonstrated normal expression of FDX2 mRNA but severely reduced expression of FDX2 protein in muscle tissue. This study describes a novel complex neurological phenotype with unusual MRI and muscle biopsy features, conclusively mapped to a mutation in FDX2, which encodes a ubiquitously expressed mitochondrial ferredoxin essential for early [Fe-S] cluster biogenesis. This study was performed by several members of our CEPID, associated to international contributers, in a publication in the journal BRAIN *(Gurgel-Giannetti J, et al , Brain, 2018).*

Manifesting carriers in recessive X-linked myotubular myopathy

Myotubular myopathy is a rare genetic disease which affects skeletal and respiratory muscles, and is caused by mutations in the *MTM1* gene. The disease is classified as recessive X-linked, i and manifests in living born males with an estimated incidence of 1/50,000. Myotubular myopathy is characteristic and very severe, including hypotonia and generalized muscle weakness since birth. Most patients die in the first year of life due to respiratory failure. However, many patient with a more benign phenotype have been recently identified

through molecular analysis. Women carrying the mutations are usually asymptomatic, but many symptomatic heterozygous females have been reported, as compared with the lower frequency of manifesting carriers in other X-linked recessive diseases. Mutations in the *MTM1* gene were identified in patients from twelve different families, using a NGS panel for neuromuscular disorders. Seven among these mutations were novel. In two families, we identified 4/8 and 2/4 female carriers presenting some degree of clinical manifestation. Adding these cases and others from the literature, we estimated the penetrance rate of 31.5% in females, which is compatible with a pattern of incomplete penetrance, and could explain the higher frequency of manifesting women. A NGS exome study is ongoing to try to identify possible modifier genes to explain this clinical variability. Souza, L. S., Almeida, Vainzof, M. MSc Dissertation. Presented and selected for highlight presentations in the World Muscle Society Meeting 2018. Received Elsevier Award, 2018

X-linked myopathy with excessive autophagy (XMEA)

X-linked myopathy with excessive autophagy (XMEA) is an inherited, slowly progressive myopathy, characterized by sarcoplasmic vacuoles in muscle fibers. XMEA is caused by mutations in the vacuolar membrane ATPase 21 gene (*VMA21*), resulting in a reduction of both mRNA and VMA21 protein levels, elevating lysosomal pH, partially blocking the final degradation step of autophagy and increasing the formation of autolysosomes. We recently identified a Brazilian family with XMEA caused by a unique small insertion-deletion in the Vma21 gene.We studied immortalized myoblasts, isolated from muscle biopsies from one XMEA patient and one control. Through qPCR, the relative expression of autophagy-related genes was evaluated, and Myog analysis confirmed myotube formation. In controls, the autophagy genes Lc3b, Vps34 and Atg12 showed a similar pattern of expression in undifferentiated cells as well as after myotube differentiation. Interestingly, in the XMEA patient, these genes showed a lower expression in myoblasts, but a higher expression after myotube formation, suggesting a more activated autophagic gene induction. Immunofluorescence analysis using anti-LC3 antibody confirmed the gene expression data at the protein levels . Our results show that the increase

in autophagy that characterizes XMEA may arise after cells start to differentiate, and that in progenitor cells such as myoblasts, the signaling may be affected in a distinct way. Stephanie de Alcantara Machado, MSc thesis. Manuscript in preparation.

A2.2. *Craniofacial disorders*

Richieri-Costa-Pereira Syndrome (RCPS): insights in the origin of the disease and microcephaly is part of the phenotype.

In 2014, we demonstrated that the excess of repeats at the 5´UTR of *EIF4A3*, a gene involved in the basic cell control of splicing and translation, causes a rare craniofacial disorder - RCPS (Favaro et al., 2014). In order to better understand the origin of these repeats and their impact on RNAm transcription, we analysed the genetic structrure of the 5´UTR in 360 control individuals and defined that the large number of repeats very likely arised by unequal crossing over, and has occurred more than once. We also showed a positive correlation between the number and sequence of the repeats with transcription levels (Hsia et al., 2018). In order to better delineate the spectrum of clinical variability of RCPS, in collaboration with Dr. Bertola, a CEPID researcher, we studied other RCPS patients and observed that microcephly, which has not been previously described in RCPS, appears as a clinical sign of the syndrome, thus expanding the complexity of the phenotype *(Bertola et al., 2018).*

A2.3. *Neurodegeneration*

Intracellular trafficking and protein aggregation in neurodegeneration

During the last year we demonstrated that treadmill running practiced before or after the beginning of neurodegeneration may protect motor cortex neurons, whereas prolonged mild running seems to be beneficial for spinal cord in terms of oxidative stress, protein aggregation and activation of autophagy (Melo, et al., 2018). Moderate physical training also could prevent early neurodegeneration in substantia nigra through the improvement of autophagy and mitophagy (Almeida, et al., 2018). In a cellular model of neurodegeneration,

using the exogenous expression of amyloid-beta peptide, we showed that proteasomal stress contributes to Alzheimer's disease-type pathogenesis and that governing such pathology occurs through crosstalk between proteasome and autophagy pathways *(Farizzato, et al., 2017).*

A3. Epigenetics and diseases

A3.1. *DNA methylation in congenital disorders*

Prader-Willi and syndromes associated with obesity (syndromic obesity)

Chromosomal microarray analysis in 279 patients with syndromic obesity (obesity with additional phenotypes) was performed and reveals multiple recurring disease-causing CNVs and novel obesity-risk loci *(D'Angelo et al., 2018).* Whole-exome sequencing analyses of the unresolved cases are in progress.

Twin girls with an atypically severe PWS phenotype were reported on whom combined analysis of the clinical features and molecular studies identified a blended phenotype likely explained by a dual molecular diagnosis of Prader-Willi and Pitt-Hopkins syndrome *(Jehee et al. 2017).*

Chromosomal microarray analysis in 279 patients with syndromic obesity (obesity with additional phenotypes) was performed and revealed multiple recurring disease-causing CNVs and novel obesity-risk loci. The CNVs detected affect several currently known candidate genes, such as *HDAC4*, *MYT1L*, *SIM1*, *POU3F2*, *EHMT1*, *SH2B1*, and *RAI1*. We also proposed novel candidates, for instance *TAS1R3*, *GAS6*, *ALOX5AP*, and *SGCG*/*MIPEP*. In addition, three relatively small CNVs with uncertain significance affected genes previously mapped to CNV loci detected in patients with syndromic obesity (*PLIN2*, *LINGO2* and *MACROD2*).These results underscore that the locus heterogeneity in syndromic obesity due to diagnosis for patients can be challenging. CMA provides an important diagnostic value and may help defining new rare genetic forms of obesity . Our next step will be whole-exome sequencing analyses of the unresolved cases.

Epigenetics in NSCLP

Epigenetic studies allow the investigation of the impact of environmental factors in the human genome and its correlation with disease susceptibility. In this context, we considered evaluating the possibility of an epigenetic signature in NSCLP, a complex disorder with high heritability, but still with non-understood genetic contribution to its etiology. We, therefore, performed amethylome-wide association study in Brazilian NSCLP (n=67) and controls (n=59), using DNA from peripheral blood. This study revealed 578 methylation variable positions significantly associated with NSCLP, further validated in a different population of European ancestry and in different tissues. We next tested if methylation could contribute to penetrance of the phenotype in individuals heterozygous forLoF variants in *CDH1*. We observed that methylation levels were significantly higher in penetrant *CDH1*/LoF NSCLP individuals, as compared to non-affected individuals and non-affected carriers of *CDH1*/LoF variants *(*Alvizi et al., 2017*).* In summary, epigenetics play a role in NSCLP and in a proportion of cases, the penetrance of the NSCLP is the result of pathogenic rare LoF variants combined with epigenetic changes, which in turn, were possibly triggered by environmental factors that the fetus were exposed during pregnancy.

A3.3. Epigenetic signature of differentially methylated genes in cutaneous melanoma

Epigenetic dysregulation is an important emerging hallmark of cancer origin and development. Cutaneous melanoma (CM) is the most aggressive subtype of skin cancer, with increasing incidence over the past several decades. In order to understand the relationship of DNA methylation in CMs, we searched for an epigenetic signature of cutaneous melanomas, by comparing the DNA methylation profiles between tumours and benign melanocytes, the precursor cells of CM. A signature of 514 differentially methylated genes (DMGs) was evident in CMs compared to melanocytes, which was independent of the presence of driver mutations. Pathway analysis of this CM signature revealed an enrichment of proteins involved in the binding of DNA regulatory regions

(hypermethylated sites), and related to transmembrane signal transducer activities (hypomethylated sites). The methylation signature was validated in an independent dataset of primary CMs, as well as in lymph node and distant metastases (correlation of DNA methylation level: r > 0,95; Pearson's test: p < 2.2e-16) *(Pramio et al, 2017).*

A3.4. How DNA damage and Genome Instability can be implicated in human disease?

During this last period, we have worked on how UVA-light (315-400 nm, the main UV component of sunlight) can damage DNA and promote biological effects in human cells. Although extensively studied, gaps in the knowledge are clear, and we decided to use cells from xeroderma pigmentosum patients (deficient on DNA damage repair or tolerance), in order to unveil these effects *(Schuch et al*, *2017).* These experiments were initially done in XP-V cells (deficient on the translesion synthesis, TLS, DNA polymerase eta). The results clearly indicated that, although oxygen radicals correspond to a late UVA-effect (not directly due to photosensitization of cell components), they play important roles in the damaging action of this light. Also the ATR/CHK1 pathway is strongly activated in these cells, protecting from deleterious UVA-effects. These results reveal not only how XP-V patients may suffer from UVA-light, but also as the skin of the human population, in general, is affected, with severe implications for carcinogenesis and skin aging (Moreno et al, 2018). In addition, a full review on the mechanisms of TLS in human cells was published *(Quinet, Lerner et al, 2018)*.

We also identified a novel mutation in the *LMNB1* gene, in a patient suffering from autosomal dominant leukodystrophy, which include neuropathological clinical phenotype. The results clearly indicate that the effect in the structure of the nuclear envelope is also responsible for genetic instability after cell treatment with topoisomerase inhibitors, in a mechanism that maybe related to the patient's phenotype *(Pedroso et al, 2017)*.

B. THE 80plus PROJECT

SABE and 80plus whole genome sequence dataset

This project was initiated aiming to have a database from a cohort of elderly individuals from the Brazilian population. The census-based elderly cohort of São Paulo city sampled from the Healthy, Well being and Aging Study (SABE – Saúde, BemEstar e Envelhecimento) was whole genome sequenced along with the cognitively healthy octogenarians '80plus' sample. A first exome analysis of 609 subjects from this cohort was recently published *(*Naslavsky et al., 2017*),* and deposited in a web-based public database ABraOM (Online Archive of Brazilian Mutations).

Preliminary results from a combined dataset of 1,172 unrelated individuals from the SABE cohort has yielded more than 78 million variants, including single nucleotide variants and short insertions and deletions. Among those, 6.7 million variants were absent from large public datasets such as gnoMAD and dbSNP (release version 150). Focusing on the 59 actionable genes recommended by the American College of Medical Genetics and Genomics (ACMG), in which pathogenic variants should be reported back to sequenced patients and subjects, we have found 123,371 variants, of which 186 have potential loss of function consequences, all individuals carrying three up to 17 such variants. Among these, 126 variants present a gnoMAD database population frequency of less than 0.1% or are absent from public databases; 196 individuals carry at least one loss of function mutation in these gene secondary findings. Further pathogenicity analyses and investigation of co-segregation of these variants and affected phenotypes is necessary to establish the clinical relevance of these findings. These results were presented in the 2018 ASHG meeting in San Diego, and a manuscript will be submitted.

In addition, whole genome sequences from the 1,172 subjects, which represent the largest cohort from Latin America, were transferred to our CEPID high performance computers. These data opened the possibility of several ongoing collaborations with different groups such as:

a) identification of retroelements (RNA retrocopies, mRNAs, L1, Alus and

LTRs), Collaboration with Dr. Pedro Galante from Hospital Sirio-Libanês:

b) analysis of DNA repair in healthy nonagenarians as compared to patients with conditions caused by defective DNA repair. Collaboration with Prof. CF Menck from ICB -USP

c) mitochondrial analysis in healthy versus unhealthy individuals older than 60 yrs. Collaboration with Prof. Anibal Vercesi from UNICAMP

d) analysis of local ancestry. Collaboration with Prof. Diogo Meyer and Regina Mingroni-Netto.

C. THERAPIES IN GENETIC DISORDERS

C1. Human stem cells

Gene editing in blood derived human induced pluripotent stem cells

Stem cells derived from patients have been extremely useful to reveal new pathogenic mechanisms and point out new targets for therapies. On this respect, CRISPR-Cas9 editing has been a powerful tool to evaluate the effects of mutations *in vitro* .An efficient one-step procedure to reprogram fibroblasts into human induced pluripotent stem cells (hiPSC), and perform CRISPR/Cas9 gene editing simultaneously was recently reported. We show that such simultaneous reprogramming and gene editing can be efficiently done with erythroblasts *(Melo et al., 2018).* We successfully obtained human induced pluripotent stem cell colonies together with *in frame* and *out of frame CAPN1* mutations in one or both alleles. We did not identify off-targets in edited cell lines. The entire process, from blood collection to mutated hiPSC took approximately five weeks, a much shorter period than standard multi-step methodologies using fibroblasts. Since blood drawing is a less invasive procedure than a skin biopsy it will allow us to investigate a significant larger number of individuals of scientific interest.

Down Syndrome iPSC-Derived Astrocytes Impair Neuronal Synaptogenesis and the mTOR Pathway In Vitro

Several methods have been used to study the neuropathogenesis of Down syndrome (DS), such as mouse aneuploidies, post mortem human brains, and in vitro cell culture of neural progenitor cells. More recently, induced pluripotent stem cell (iPSC) technology has offered new approaches in investigation, providing a valuable tool for studying specific cell types from individuals with DS, especially neurons and astrocytes. Here, we investigated the role of astrocytes in DS and the impact of the astrocyte secretome in neuron mTOR signaling and synapse formation, using iPSC derived from DS and wild-type (WT) subjects. We demonstrated, for the first time, that DS neurons derived from hiPSC recapitulate the hyperactivation of the Akt/mTOR axis observed in DS brains, and that DS astrocytes may play a key role in this dysfunction. Our results bear out that 21 trisomy in astrocytes contributes to neuronal abnormalities in addition to cell autonomous dysfunctions caused by 21 trisomy in neurons. Further research in this direction will likely yield additional insights, thereby improving our understanding of DS and potentially facilitating the development of new therapeutic approaches *(Araujo et al., 2018)*

MSCS secretome characterization

Despite several advances, there is no effective therapy for Duchenne Muscular Dystrophy (DMD). Therefore, the potential regenerative capacity, and immune-privileged properties of mesenchymal stromal cells (MSCs), have been the focus of intense investigation in different animal models, aiming at the treatment of DMD. We analyzed the protein content of the secretome of MSCs, isolated from three different sources (adipose tissue, skeletal muscle, and uterine tubes), obtained from five donors and evaluated their *in vitro* properties, when co-cultured with DMD myoblasts. All MSC lineages showed pathways enrichment related to protein metabolic process, oxidation-reduction process, cell proliferation, and regulation of apoptosis. We found that MSC secretome proteins and their effect *in vitro* vary significantly according to the tissue and donor, indicating the importance of characterizing MSC secretome profile before its use in animal and clinical trials. Despite the individual differences a pool of

conditioned media from all MSC lineages was able to delay apoptosis and enhance migration, when in contact with DMD myoblasts. Interestingly, in the co-culture of one donor's secretome, in which we observed a more significant delayed apoptosis in contact with DMD myoblasts, there was an increased expression of several proteins including JAGGED-1. This finding called our attention since overexpression of jagged-1 was identified as responsible for the mild phenotype and normal life reported in two golden-retriever (GRMD) dogs from our colony. This work was the MSc thesis of Amanda Assoni (MayanaZatz, supervision) and was published in Stem Cells Dev (Assoni et al., 2017).

Human Adipose-Derived CD146$^+$ Stem Cells increase life span more efficiently than Mesenchymal Stromal Cells

Duchenne muscular dystrophy is the most common and severe form of progressive muscular dystrophies. Previous results showed an increased survival in double knockout mice (dko), when treated with adipose-derived CD146$^+$ cells. In this study, we analyzed the effect of CD146$^+$ cells compared to mesenchymal stem/stromal cells (MSCs) derived from the same human adipose sample, when injected in the dko mouse model without immunosuppression. Both CD146$^+$ cells and MSCs increased the survival of treated mice, when compared to vehicle-injected mice, with a more prominent effect of CD146$^+$ cells than MSCs. Both CD146$^+$ cells and MSCs suppressed peripheral blood mononuclear cell proliferation, indicating immunomodulatory properties. Co-culture experiments showed that MSCs have a more inflammatory profile expression, and angiogenesis assay showed that CD146$^+$ cells can improve blood vessel formation. CD146$^+$ cells can extend survival of muscular dystrophy mice more efficiently than MSCs, possibly due to immunomodulatory and angiogenic properties. Further investigations focusing on exogenous CD146$^+$ cell role *in vivo* will improve cell therapy understanding and effectiveness *(Gomes et al., 2018).*

Human Pericytes Extend Survival of ALS SOD1 Mice

Amyotrophic Lateral Sclerosis (ALS) is one of the most common adult-onset motor neuron disease, causing a progressive, rapid and irreversible

degeneration of motor neurons in the cortex, brain stem and spinal cord. No effective treatment is available and cell therapy clinical trials are currently being tested in ALS affected patients. It is well known that in ALS patients, approximately 50% of pericytes from the spinal cord barrier are lost. In the central nervous system, pericytes act in the formation and maintenance of the blood-brain barrier, a natural defense that slows the progression of symptoms in neurodegenerative diseases. We evaluated, for the first time, the therapeutic effect of human pericytes *in vivo*, in SOD1 mice, and *in vitro*, in motor neurons and other neuronal cells derived from one ALS patient. Pericytes and mesenchymal stromal cells (MSCs) were derived from the same adipose tissue sample and were administered to SOD1 mice intraperitoneally. The effect of the two treatments was compared. Treatment with pericytes extended significantly animal survival in SOD1 males, but not in females that usually have a milder phenotype with higher survival rates. No significant differences were observed in the survival of mice treated with MSCs. Gene expression analysis in brain and spinal cord of end-stage animals showed that treatment with pericytes could stimulate the host antioxidant system. Additionally, pericytes induced the expression of *SOD1* and *CAT* in motor neurons and other neuronal cells derived from one ALS patient carrying a mutation in *FUS*. Overall, treatment with pericytes was more effective than treatment with MSCs. Our results encourage further investigations and suggest that pericytes may be a good option for ALS treatment in the future. (*Coatti et al., 2017)*

Mechanisms that confer increased osteogenic potential in stem cells

One of our goals is to identify factors that would confer an increased osteogenic potentail in mesenchymal stem cells (MSCs). To achieve this goal, we have characterized *CD105* expression and its regulation in MSCs from different tissues with different osteogenic potential, for example, MSCs from exfoliated dental tissue (SHED) as compared to MSC from adipose tissue (hASD). We showed that *CD105* in SHED, which presents a higher osteogenic potential than hASD, is regulated by a micro-RNA, and could in turn become a tool to be used to improve *in vivo* bone regeneration. *(Ishiy et al., 2018)*.

C2. Pre-Clinical studies with murine stem cells

Muscle satellite cells and impaired late stage regeneration in different murine models of muscular dystrophies

Satellite cells (SCs) are the main stem cells of the muscle, responsible for its regenerative capacity after injury. In muscular dystrophies, SCs are constantly activated, but a failure of the regenerative process results in muscle degeneration and weakness. We studied muscle SCs in three mouse dystrophic strains: DMDmdx, Largemyd, DMDmdx/Largemyd, to evaluate SCs behavior in muscles with different degrees of degeneration. The dystrophic muscles from the three strains showed similar results, retaining satellite cell pool, expressing PAX7, an important muscle factor for self-renewal of the SC pool. Expression analysis demonstrated that the cascade of regeneration genes was also activated in all the dystrophic muscles, with high levels of MYOD and Myogenin. The ability to form new fibers was also preserved, with the presence of a significant number of new fibers expressing dMHC. However, these new fibers show incomplete maturation characteristics, such as small size and no variation in fiber caliber, which could be determinant for its dysfunction. On the other hand, muscle degeneration was intense, with significant more connective tissue infiltration in dystrophic mice. We concluded that dystrophic muscles, independently of the degree of degeneration, retain the pool of satellite cells with proliferating capacity and ready to respond to regenerating stimuli. However, the maturation of these new fibers is incomplete and do not prevent the degeneration of the muscle. Efforts to improve late muscle regeneration should better contribute to therapeutic approaches. Antonio F. Ribeiro Junior,. MSc Dissertation; supervisor: Mariz Vainzof Presented in the meeting of the World Muscle Society, and received the Elsevier Award 2018. The manuscript was submitted for publication.

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C3. Safety-related concerns in cell therapy

During this period we also have published two reviews, indicating how DNA repair and autophagy may protect tumor cells from DNA damaging agents, and make the tumors resistant to therapy *(Gomes et al, 2017; Rocha et al, 2018)*.

One of them addresses the question whether MSCs can be used to treat cancer. In cancer, mesenchymal stem/stromal cells (MSCs) have been considered as vehicles for targeted delivery of drugs due to their inherent tropism toward primary and metastatic tumors. However, it is still unclear whether MSCs could be therapeutically explored without significant harm, since a great amount of evidence indicates that MSCs are able to exert both tumor-suppressive and pro-oncogenic effects. In an attempt to address this question we discuss how MSCs might adopt a pro- or an anti-inflammatory profile in response to changes within the tumor microenvironment, and how these features may lead to opposite outcomes in tumor development. Additionally, we address how differences in experimental design might impact interpretation and consistency of the current literature in this specific field. Finally, we point-out critical issues to be addressed at a pre-clinical stage, regarding safety and therapeutic effectiveness of MSCs application in cancer treatment. *(Gomes et al., 2017)*

Indeed, studies concerning the effects of mesenchymal stem cells (MSC) on the tumor microenvironment (TME) found that MSC are capable of stimulating human Glioblastoma (GBM) cell proliferation through a paracrine effect mediated by TGFB1. When in direct cell-cell contact with GBM cells, MSC elicited an increased proliferative and invasive tumor cell behavior under 3D conditions, as well as accelerated tumor development in nude mice, independently of paracrine TGFB1. A secretome profiling of MSC-GBM co-cultures identified 126 differentially expressed proteins and 10 proteins exclusively detected under direct cell-cell contact conditions. Most of these proteins are exosome cargos and are involved in cell motility and tissue development. These results indicate a dynamic interaction between MSC and GBM cells, favoring aggressive tumor cell traits through alternative and independent mechanisms. We have also performed a comparative characterization of normal and tumoral pericytes (isolated from childhood ependymoma and neuroblastoma specimens) and reported for the first time the modulatory effects of LOX enzymes on activated pericyte properties. In most pericyte samples, LOXL3 was the family member displaying the highest transcript levels. Inhibition of LOX/LOXL activity with the inhibitor β-

aminopropionitrile (βAPN) significantly reduced migration of pericytes, while proliferation rates were kept unaltered. Formation of tube-like structures in vitro by pericytes was also significantly impaired upon inhibition of LOX/LOXL activity with βAPN, which induced more prominent effects in tumor-associated pericytes. These findings reveal a novel involvement of the LOX family of enzymes in migration and angiogenic properties of pericytes, with implications in tumor development and in therapeutic targeting tumor microenvironment constituents. Overall, these findings indicate that MSC and pericytes may exert pro-tumorigenic effects when in close contact with tumor cells, which must be carefully considered when employing these cells in cell therapy protocols.

C4. Other therapeutic approaches

Immunoglobulin therapy ameliorates the phenotype and increases lifespan in dystrophin-utrophin double knockout mice

Duchenne muscular dystrophy (DMD) is an X-linked recessive disorder, caused by mutations in the dystrophin gene, affecting 1:3,500-5,000 boys worldwide. The lack of dystrophin induces degeneration of muscle cells and elicits an immune response characterized by an intensive secretion of pro-inflammatory cytokines. Immunoglobulins modulate the inflammatory response through several mechanisms and have been widely used as an adjuvant therapy for autoimmune diseases. We evaluated the effect of immunoglobulin G (IG) injected intraperitoneally in a severely affected double knockout (dko) mouse model for Duchenne muscular dystrophy. The IG dko treated mice were compared with a control untreated group, regarding activity rates, survival and histopathology. Additionally, dendritic cells and naïve lymphocytes from these two groups and WT mice were obtained to study *in vitro* the role of the immune system associated to DMD pathophysiology. We showed that IG therapy significantly enhanced activity rate and lifespan of dko mice. It diminishes muscle tissue inflammation by decreasing the expression of costimulatory molecules MHC, CD86 and CD40, and reducing Th1-related cytokines IFN-γ, IL-1β and TNF-α release. IG therapy dampens the effector immune responses supporting the hypothesis according to which the immune response accelerates

DMD progression. As IG therapy is already approved by FDA for treating autoimmune disorders, with less side-effects than currently used glucocorticoids, our results may open a new therapeutic option aiming to improve life quality and lifespan of DMD patients.*(Nunes et al., 2017).*

Efficient exon skipping of SGCG mutations mediated by morpholino oligomers

Exon skipping uses chemically modified antisense oligonucleotides to modulate RNA splicing. Therapeutically, exon skipping can bypass mutations and restore reading frame disruption by generating internally truncated, functional proteins to rescue the loss of native gene expression. Limb-girdle muscular dystrophy type 2C is caused by autosomal recessive mutations in the *SGCG* gene, which encodes the dystrophin-associated protein γ-sarcoglycan. The most common *SGCG* mutations disrupt the transcript reading frame abrogating γ-sarcoglycan protein expression. In order to retore the SGCG transcript reading frame in most *SGCG* gene mutations, it is necessary to skip four exons, creating an internally truncated protein referred to as Mini-Gamma. Using direct reprogramming of human cells with MyoD, myogenic cells were tested with two antisense oligonucleotide chemistries, 2'-O-methyl phosphorothioate oligonucleotides and vivo-phosphorodiamidate morpholino oligomers, to induce exon skipping. Treatment with vivo-phosphorodiamidate morpholino oligomers demonstrated efficient skipping of the targeted exons and corrected the mutant reading frame, resulting in the expression of a functional Mini-Gamma protein. Antisense-induced exon skipping occurred in normal cells and those with distinct SGCG mutations, including the most common 521ΔT mutation. These findings demonstrate a multiexon-skipping strategy applicable to the majority of limb-girdle muscular dystrophy 2C patients *(Wyatt et al., 2018)*.

Zika Virus Selectively Kills Aggressive Human Embryonal CNS Tumor Cells *In Vitro* and *In Vivo*.

Zika virus (ZIKV) is largely known for causing brain abnormalities due to its ability to infect neural progenitor stem cells during early development. Here, we show that ZIKV is also capable of infecting and destroying stem-like cancer cells from aggressive human embryonal tumors of the central nervous system (CNS). When evaluating the oncolytic properties of Brazilian Zika virus strain ($ZIKV^{BR}$) against human breast, prostate, colorectal, and embryonal CNS tumor cell lines, we verified a selective infection of CNS tumor cells followed by massive tumor cell death. $ZIKV^{BR}$ was more efficient in destroying embryonal CNS tumorspheres than normal stem cell neurospheres. A single intracerebroventricular injection of $ZIKV^{BR}$ in BALB/c nude mice bearing orthotopic human embryonal CNS tumor xenografts resulted in a significantly longer survival, decreased tumor burden, fewer metastasis, and complete remission in some animals. Tumor cells closely resembling neural stem cells at the molecular level with activated Wnt signaling were more susceptible to the oncolytic effects of $ZIKV^{BR}$Furthermore, modulation of Wnt signaling pathway significantly affected $ZIKV^{BR}$-induced tumor cell death and viral shedding. Altogether, these preclinical findings indicate that $ZIKV^{BR}$ could be an efficient agent to treat aggressive forms of embryonal CNS tumors and could provide mechanistic insights regarding its oncolytic effects. Tumor cells closely resembling neural stem cells at the molecular level with activated Wnt signaling were more susceptible to the oncolytic effects of Zika. These novel findings were published in Cancer Research and highlighted in its cover (Kaid et al., 2018). This study was also recently mentioned in a spotlitght article in Nature, about the scientific reseach scenario in São Paulo *(Nature. 2018 Nov;563 (7733):S179-S181.).*

PART 2
TRANSFER OF TECHNOLOGY/TECHNOLOGY APPLICATIONS

As transfer of technology, our proposal is to translate scientific and technological advances into services, as follows:

a) Sequencing Facility (EMU/ Equipamento Multiusuário /Multiuser Equipment-FAPESP): HUG-CEL EMU (http://genoma.ib.usp.br/servicos) contains three sequencing apparatus (ABI 3730 DNA Analyser sequencer (Applied Biosystems), MiSeq and HiSeq 2500 (Illumina) and infrastructure for storage and data processing (total storage capacity of 660 TB with 60 TB allocated at USP Cloud and two processing servers with 512 GB RAM and 32 cores in total). This is a result of a 2016 expansion, with the acquisition of a storage server with 480 TB partially financed by USP. We also set up the Bravo robot, which is being used for exome library preparations. The managing committee is meeting once a year, while the User committee constantly provides suggestions to improve organization http://genoma.ib.usp.br/servicos/sequenciamento-de-nova-geracao-ngs/comite). A total of 632 NGS tests (531 paid; 101 research) and ~ 48,632 sanger sequencing/microsatellite reactions (13,632 research; 35,000 paid) were performed at HUG-CEL EMU for 9 researchers, about 250 external users and samples for genetic diagnosis from our non-profit laboratory.

b) Bio-repository: A collection of more than 20,000 DNA samples of patients with genetic disorders and their relatives has been established in the last 30 years. In addition to somatic cell cultures (fibroblast, myoblasts), we have established induced pluripotent stem cells (iPSC) of 121 with different genetic disorders and controls in the last 6 years. Most of these cell lines are being used by CEPID researchers.

c) Genetic counseling service: About 1274 families (2012 consultations) were attended by our team (about 80% at the HUG-CEL and the remaining ones in other hospitals in Sao Paulo or in other regions of Brazil). Genetic counseling

of families with affected patients includes diagnosis, identification and testing of “at-risk carriers”, orientation about prognosis and management and genetic counseling. Written reports were provided to all attending individuals.

d) Genetic Tests: The web page of the non-profit laboratory for genetic tests (http://laboratorio.genoma.usp.br) is being constantly updated with the inclusion of new tests, as for example the new NGS test that analyses 6700 genes associated with rare diseases. During the last year, we have performed 1068 paid genetic tests (MLPA/disease specific CNVs, Triple/PCR for expansion, NGS panels, NGS mini-exome, NGS exome). The quality and reliability of our genetic tests have been certified yearly by the European Molecular Genetics Quality Netwok (EMQN). Additionally, about 609 tests (Sanger sequencing, MLPA, NGS, Cytogenetics- karyotype, array-comparative genomic hybridization) related to research projects were also performed by HUG-CEL. The sequencing reactions of these tests were performed by the EMU facility. Non-invasive prenatal diagnosis was established (Malcher et al., 2018) and we have been discussing about the feasibility to implement it or not.

e) DATABASES: We have developed, and hosted in our servers, a public access website (http://abraom.ib.usp.br) to provide information on the frequency of variants in 609 Brazilian healthy individuals that are part of the Sao Paulo city elderly cohort studied at our center (SABE cohort). The data has provided valuable information for the interpretation of pathogenicity of variants identified in genetic tests in Brazil and around the world. We are now developing a new database, DesBraVar, that will include all the NGS data generated in our center (1324 whole genome sequences from elderly, 400 exome sequences from affected individuals with rare disorders and approximately 2000 NGS tests). Finally, we are updating the ZEN phenotypic database, which stores clinical data from the families with genetic disorders. We expect in the near future to integrate ZEN and DesBraVar so that queries will be possible with both genotypic and phenotypic data.

f) Income resources administration: The income of the genetic tests and

services are being used to pay for activities not supported by our current grants or university, such as payment of technicians), equipment maintenance and reagents for the genetic tests. This income is being administrated at the fundação Faculdade de Medicina USP and Fundação Universidade de São Paulo.

PART 3
EDUCATION OUT REACH

A. High School Support Program

A.1. Project: Laboratory classes at school

http://www.genoma.ib.usp.br/pt-br/educacao-e-difusao/nossos-projetos/parcerias-com-diretorias-de-ensino/aulas-praticas-nas-escolas

We establish laboratory classes within individual schools for periods of 3 weeks, where teachers were assisted in leading laboratory classes related to the cellular basis of Genetics, including the use of microscopes and 6 different practical kits *(annexes 4.1 to 4.3).* 16 hours of technical and pedagogical support to 54 High School teachers were delivered; 58 students were trained to act as monitors during the time the laboratory is installed in their schools; 94 High Schools were assisted, from August/2017 to November/2018 and nearly 66,000 students were benefited.

A.2. Instructional support project

http://www.genoma.ib.usp.br/educacao-e-difusao/nossos-projetos/parcerias-com-diretorias-de-ensino/material-instrucional-nas-escolas The objective of the project is to help teachers to over-come some of the teaching and learning difficulties presented by the abstract nature of some Genetics concepts. We provided instructional support material to facilitate the teaching and learning processes and established three loan centers, which currently provide instructional material to more than 100 teachers each year. 32 hours of technical and pedagogical support to 44 High School teachers were delivered for teachers of Biology, Sciences and Physics *(annex 4.4).*

A.3. Scientific Exhibitions

The "**Giant Cell**" http://www.genoma.ib.usp.br/pt-br/educacao-e-difusao/nossos-projetos/celula-gigante, a scenic cell amplified 130,000 times and a set of complementary activities designed to facilitate the understanding of cell concepts and a Scientific exhibition **"Light and Life" (USP goes to your school project)** http://www.genoma.ib.usp.br/pt-br/educacao-e-difusao/nossos-projetos/parcerias-com-diretorias-de-ensino/usp-vai-a-sua-escola_were visited by 7,700 people in this period *(annex 4.5).*

B. Project having patients and their families as target

B.1. Educational leaflets –six more leaflets of a series of 12 educational leaflets were produced in the period.

http://www.genoma.ib.usp.br/educacao-e-difusao/materiais-didaticos/folhetos

C. Projects having the general public as target

YouTube channel and Facebook account

https://www.youtube.com/channel/UCKoucKINM7-LNaR3grrMcYw

https://www.facebook.com/pordentrodogenoma/. We produce videos about genetics for the general public that are uploaded to our YouTube channel and Facebook account every week. We have developed three video formats: *Genomic News*, with news about the research at HUG-CELL; *Speak Out, Geneticist!*, in which researchers are interviewed and talk about a variety of issues related to genetics; and *Laboratory Life*, which is produced by graduate students and show what the everyday life at the lab is like, including curious or funny aspects. The number of followers on Facebook has been rising steadily, and we now count with nearly 1,600 followers (from 500 in April).

D) Interviews to the Media and Science Dissemination Articles

The interaction with the media to discuss, translate and disseminate new scientific discoveries to lay people was achieved through 136 interviews and articles of science dissemination. *(annex 4.6)*

Annex 1
Publications in peer reviewed journals, books and patent

From July 2017 until November 2018, our group has published 97 journal articles (all listed below), 2 books or book chapters, 22 abstracts in National meetings, and 42 abstracts in International meetings. During this period, our graduate students submitted 7 Master Theses and 5 Doctoral Dissertations. About 37 conferences, lectures and symposia were done by our time.

1. Articles

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2. Books and Book Chapters

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2. **Ferrari MFR**. Intracellular Transport System in AD. In: Fernando A. Oliveira. (Org.). Recent Advances in Alzheimer's Research: Cellular mechanisms in Alzheimer's Disease. 1ed.: Bentham, 2018, v. 2, p. 140-160.

3. Patents

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Annex 2

Meetings, Conferences, Lectures

1. Abstracts: National Meetings

1. Andreis TAF, **Ferrari MFR**. Analysis of Miro-1 upon cell viability in alpha-synuclein overexpressing cells. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.
2. Avelino CC, Pinheiro MMLS, **Vibranovski MD**. “Drosophila melanogaster spermatogenic stage-specific expression: quantity and quality evaluation for next generation sequencing”, X Simpósio de Ecologia, Genética e Evolução de Drosophila, Hotel SESC Estalagem Ouro Preto. Ouro Preto, MG, Brasil, 2017
3. Barbieri BD, **Okamoto OK**. Role of glutathione depletion in chemotherapy resistance in Aggressive Medulloblastoma. In: 63ª Reunião Anual da Sociedade Brasileira de Genética, 2017, Águas de Lindoia. Abstract compilation book, 2017.
4. Cardoso RR, Araujo FT, Pereira LV, **Ferrari MFR**. Autophagy flux in hiPSC-derived dopaminergic neurons from Parkinson´s and Gaucher´s disease patients. In: Fesbe Regional, 2018, Rio Grande. XII Reunião Regional da FeSBE, 2018.
5. Cardoso RR, **Ferrari MFR**. Overexpression of Lamp-1 prevents alpha-synuclein accumulation in locus coeruleus. In: XIX Congress of the Brazilian Society for Cell Biology, 2018, Sao Paulo. XIX Congresso da SBBC, 2018.
6. Cardoso RR, **Ferrari MFR**. Analysis of autophagy flux during protein aggregation in cultured cells from hippocampus. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.
7. Domingos RM, Teixeira RD, Zeida A, Alegria TGP, Estrin D A, **Netto LES**. First Crystallographic Structure of the Interaction Between an Organic Hydroperoxide Resistance Protein and its Biological Reductant: Structural and Molecular Dynamic Analyses In: 46a Reunião Anual da SBBq, 2017, Águas de Lindóia. 46a Reunião Anual da SBBq. , 2017. p.E-27 Referências adicionais : Brasil/Português.
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10. Lima RS, Silva CM, **Ferrari MFR**. Effects of physical exercise upon proteostasis during early neurodegeneration. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.

11. Melo KP, **Ferrari MFR**. Analysis of mitophagy during protein aggregation associated with neurodegeneration of substantia nigra during neurodegeneration. In: XIX Congress of the Brazilian Society for Cell Biology, 2018, Sao Paulo. XIX congresso da SBBC, 2018.
12. Melo KP, Silva CM, **Ferrari MFR**. Effects of moderate treadmill running on mitophagy in the hippocampus and substantia nigra of elderly rats during neurodegeneration. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.
13. Oliveira BS, **Ferrari MFR**. . Effects of aging and physical activity on C9orf72 expression in the hippocampus during neurodegeneration. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.
14. Price LC, Cortez, B.A, **Okamoto OK**. Study of the hippo pathway in medulloblastoma: inhibition of yap and its relation to cancer stem-cells. In: 63ª reunião anual da Sociedade Brasileira de Genética, 2017, Águas de Lindoia. Abstract compilation book, 2017.
15. Ragnoni EG, Meireles DA, **Netto LES**. Functional characterization of YmaD an Ohr/OsmC from Bacillus subtillis In: 46a Reunião Anual da SBBq, 2017, Águas de Lindóia. 46a Reunião Anual da SBBq. SBBq, 2017. p.O-14 - Referências adicionais : Brasil/Inglês. Meio de divulgação: Vários
16. Raíces J, Otto P, **Vibranovski MD**. Resolving differences on the chromosomal distributions of Drosophila new genes, X Simpósio de Ecologia, Genética e Evolução de Drosophila, Hotel SESC Estalagem Ouro Preto. Ouro Preto, MG, Brasil, 2017.
17. Ramos A.; Gomes F, **Netto LES**, Barros MH.. Roles of Prx1 from Saccharomyces cerevisiae in the mitochondrial matrix and intermembrane space In: 47th Reuniao Anual SBBq, 2018, Joinville SC. SBBq. SBBq, 2018. p.O-09 - Referências adicionais : Brasil/Inglês. Meio de divulgação: Meio digital
18. Reis JA, **Ferrari MFR**. Cellular distribution and colocalization of C9orf72 with Rab7 protein in a neurodegeneration model. In: XIX Congress of the Brazilian Society for Cell Biology, 2018, Sao Paulo. XIX Congresso da SBBC, 2018.
19. Sakugawa AYS, Queiroz EO, Andreis TAF, **Ferrari MFR**. Silencing of Miro-1 prevents endoplasmic reticulum stress in A53T alpha-synuclein overexpressing cells. In: XIX Congress of the Brazilian Society for Cell Biology, 2018, Sao Paulo. XIX Congresso da SBBC, 2018.
20. Santiago VF, **Netto LES**, Demasi M. Differntial proteomic analysis in the yeast Saccharomyces cerevisiae after site specific mutation of Cys residues in 20S proteasome In: 47th Reuniao Anual SBBq, 2018, Joinville SC. SBBq. , 2018. p.O-14 - Referências adicionais : Brasil/Inglês. Meio de divulgação: Meio digital.
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22. Teruel NFB, **Ferrari MFR**. Physical activity recovers mitophagy in animal model of senescence. In: FeSBE, 2017, Campos do Jordão. XXXII Reunião Anual da FeSBE, 2017.

2. Abstracts: International Meetings

1. Abu Hana AS, Sakata H, Oliveira CP, Bonadio RS, Ferrari I; Safatle HPN, Cordoba MS, Rosa MTAS; **Rosenberg C**, Freitas EL, Pogue RE, Acevedo-Poppe AC, Pic-Taylor A, Oliveira SF, Mazzeu JF. PgmNr 2969: Cytogenomic findings in Brazilian patients with OAVS. Poster ASHG San Diego 2018.
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3. Aguiar TFM, Costa C, Toledo SRC, Cypriano M, Carraro DM, **Rosenberg C**, **Krepischi AC**. Genomic studies in hepatoblastoma: insight into somatic mutations using array-cgn analysis and whole-exome sequencing, SIOP 2017, Washington –USA.
4. Almeida CF, Bitoun M, **Vainzof M**. Satellite cell alteration in DNM2-related centronuclear myopathy. WMS meeting of the Morld Muscle Society, Saint Malo, 3-7 october, 2017. Neurom. Disord. 27:S193, 2017
5. Almeida CF, Fernandes SA, Ribeiro AF, Ayub-Guerrieri D, **Vainzof M**. Muscle satellite cells. 10th International Conference on Cachexia, Sarcopenia & Muscle Wasting. Rome on 8-10 December 2017
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7. Assoni AF, Semedo-Kuriki P, Cortez B, **Zatz M**, **Okamoto OK**. VAPB overexpression enhances tumorsphere generation capacity of human Medulloblastoma cells. In: Annual Meeting of the International Society for Stem Cell Research, 2018, Melbourne, Australia. Abstract compilation book, 2018.
8. Barbieri BD, Marcola M, Rocha CRR, **Okamoto OK**. Glutathione depletion overcomes chemotherapy resistance in aggressive medulloblastoma stem-like. In: AACR International Conference – Translational Cancer Medicine, 2017, São Paulo. Abstract compilation, 2017. p. A57.
9. Barboza R, Castro PO, Lima RS, **Ferrari MFR**. SOD1G93A mouse model of ALS presents increased expression of NLRP1 and NLRP3 in cells from spinal cord. In: ASCB-EMBO Joint Meeting, 2017, Philadelphia. ASCB-EMBO, 2017.
10. Batissoco AC , Silva RS, Cruz DB, Alegria TGP, Gomes F, Oiticica JC, **Mingroni-Netto RC**, **Haddad LA**. A protein network associating connexins to the cytoskeleton In: International

Gap Junction Conference, 2017, Glasgow. International Gap Junction Conference - Glasgow 2017. Glasgow: , 2017. p.0-42 - 0-42

11. Bannitz-Fernandes R.; Godoy KF, Malavazi I, Anschau V, Caves Filho AB, Miyamoto S, **Netto LES**. Identification and Characterization of Reduction Agents of 1-Cys Peroxiredoxins from Aspergillus fumigatus, a Human Opportunistic Pathogen In: 19th biennial meeting forthe Society for Free Radical Research Internacional (SFRRI, 2018, Lisboa. 19th biennial meeting forthe Society for Free Radical Research Internacional (SFRRI. , 2018. Referências adicionais : Portugal/Inglês.
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13. Caires-Júnior LC, Goulart E, Telles-Silva K, Musso C, Kobayashi G, Assoni A, Ribeiro-Júnior A, Caldini E, **Passos-Bueno MR**, Rangel T, Raia S , Lelkes P, **Zatz M** . A new method for hepatic-specifichipsc-derived cells recellularization- International Congress of ISSCR, Australia, June 2018
14. Coatti G, Frangini M, Valadares M , Gomes J , Lima O, Cavanca N, Assoni A . Pelatti M Birbair A, Lima A, Singer J , Rocha F , ; Silva G , Mantovani M , Macedo-Souza L, **Ferrari MFR**, **Zatz M**. Pericytes Extend Survival of ALS SOD1 Mice and Induce the Expression of Antioxidant Enzymes in the Murine Model and in IPSCs Derived Neuronal Cells from an ALS Patient. In: 15th International Society for Stem Cell Research Annual Meeting, 2017, Boston. 15th ISSCR, 2017.
15. Cortez BA, Price LC, **Okamoto OK**. Investigating the rates of asymmetric cell division in medulloblastoma cancer stem cells. In: Annual Meeting of the International Society for Stem Cell Research, 2017, Boston. Abstract compilation book, 2017.
16. Demasi M, **Netto LES**, Santiago VF. Redox control of the 20 s proteasome gating: implications on the chronological life span of yeast cells In: 19th Biennial Meeting of the Society-for-Free-Radical-Research-International (SFRRI), 2018, Lisbon, Portugal Free Radical Biology and Medicine. New York, NY. 10010-1710 USA: Elsevier Science INC, 2018. v.120. p.S142 - S142 Referências adicionais : Estados Unidos/Inglês. Meio de divulgação: Vários
17. Dias AMM, Lezirovitz K, Marcolino HC, Nicastro FS, Mendes BCA, **Mingroni-Netto RC.** Novel mutation in CEACAM16 gene segregating with autosomal recessive deafness In: 11th Molecular Biology of Hearing and Deafness Conference, 2018, Gottingen. 11th Molecular Biology of Hearing and Deafness Conference. , 2018. v.p.31. p.58
18. Dias AMM, Lezirovitz K, Marcolino HC, Nicastro FS, Mendes BCA, **Mingroni-Netto RC,** Forbes J, Genesini T, Mouzat A, Bogochvol A, Castro D, Rüdiger D, Padovan E, Macedo E, Fonseca F, Andrade H, Silva H, Lise L, Dantas L, Naccache M, Valladares T, Pavanello R, **Zatz M**. Long term follow-up of neuromuscular patients and family members submitted to

psychoanalytical treatment. Neuromuscular Disorders. 27. S310, 2017 . 22nd meeting of the world muscule society, S. Malo, France, 2017.

19. Domingos RM, Teixeira RD, Zeida A, Agudelo WA, Alegria TGP, Murakami MT Estrin DA, **Netto LES**. Substrate triggred structural movements in Ohr: dihydrolipoamide accelerates the approximation of Catalytic Arg towards active site In: 19th biennial meeting forthe Society for Free Radical Research Internacional (SFRRI, 2018, Lisboa. 19th biennial meeting forthe Society for Free Radical Research Internacional (SFRRI. , 2018. Referências adicionais : Portugal/Inglês.
20. Ghirotto B, Vieira Loures F, Bueno H, E Cangussu, Goulart E, Coatti G, Caldini E, Condino-Neto A, **Zatz M**. Immunoglobulin therapy modulates the severe inflammatory progression of neuromuscular disorders. Neuromuscular Disorders 27. S192-S193. Neuromuscular Disorders.
21. Gomes F, Barros MH, Turano HG, **Netto LES**. Yeast mitochondrial Prx1 show dual mitochondrial localization by a process regulated by Mrg2 In: SfRMB 25th Annual conference, 2018, Chicago, IL , EUA. Free Radical Biology Medicine. ELSEVIER, 2018. v.128 S1. p.S84 - Referências adicionais : Estados Unidos/Inglês. Meio de divulgação: Vários. Home page: [http://https: //www.sciencedirect.com/science/article/pii/S089158491831952X] https://doi.org/10.1016/j.freeradbiomed.2018.10.192.
22. Gomes F, Palma FR, Barros MH, Tsuchida ET, Turano HG, Alegria TGP, Demasi M, **Netto LES**. Importing of peroxiredoxins to distinct mitochondrial compartments: possible impacts on physiology and pathology In: SfRBM 2017 SfRBM's 24th Annual Meeting, 2017, Baltimore MD USA. Free Radical Biology Medicine. , 2017. v.112. p.29 – 29 Referências adicionais : Brasil/Português.
23. Goulart E, Caires-Júnior LC, Melo US, Araujo B, Alvizi L, Soares-Schanoski A, Amaral M, **Zatz M**. Differential gene expression signature in neural progenitor cells from discordant twins for congenital zika syndrome. International Congress of ISSCR, Australia, June 2018.
24. Guercio AMF, Alegria TGP, Meireles DA, Truzzi DR, da Silva Neto Jr JF, Austo O, Trujillo M; **Netto LES**. Kinetic Characterization of the Redox Regulation of OhrR In: SfRBM 2017 SfRBM's 24th Annual Meeting, 2017, Baltimore MD USA. Free Radical Biology Medicine. , 2017. v.112. p.22 – 23 Referências adicionais : Estados Unidos/Inglês. Meio de divulgação: Vários
25. Gurgel-Gianneti J, Lynch D, Paiva A, Yamamoto G, Lucato L, Amorim S, Freua F, Giannetti A, Ripa B, Monti F, Ribeiro M, Van der Knaap M, Oldfors A, **Vainzof M**, Holden H, **Kok F**. Biallelic mutation in FDXIL leads to a complex phenotype: otic atrophy, reversible leukoencephalopahy, metabolic myopathy and axonal polyneuropathy. WMS meeting of the Morld Muscle Society, Saint Malo, 3-7 october, 2017. Neurom. Disord. 27:S206, 2017
26. Ishiba R, Bigot A, Ribeiro Jr AF, Mouly V, **Vainzof M**. Defective myoblast differentiation in human muscle dysferlin-deficient cells. American Society of Human Genetics - 2017, Orlando, October 17-21,02017

27. Fear J, Mahadevaraju S, Akeju M, Galletta B, Kennison J, **Vibranovski M**, Matunis E, Oliver B. Meiotic sex chromosome inactivation in the drosophila melanogaster male germ line Germ Cells - Cold Spring Harbor meetings, October, 2018.
28. Kaid C, Siqueira Bueno HM, Marçola M, **Okamoto OK**. miR-367 in diagnosis and therapy of childhood central nervous system embryonal tumors. In: Annual Meeting of the International Society for Stem Cell Research, 2018, Melbourne, Australia. Abstract compilation book, 2018.
29. Lazar M, Rocha KM, Varela, M,C, Yamamoto GL, Takahashi VN, Bagatini K, Ezquina S, Scliar, MO, Wang JYT, Pavanello R, Bulle Oliveira AS, **Zatz M**, **Vainzof M**, Passos-Bueno MR. Synergistic effect of mutations in dystroglycanopathies-associated genes? A Brazilian case report. American Society of Human Genetics, San Diego, October 16-20,2018
30. Naslavsky MS, Yamamoto GL, Ezquina SAM, Duarte YAO, **Passos-Bueno MR**, **Zatz M**. Cancer mutations in healthy admixed elderly: can we improve pathogenicity interpretation? International Congress of ASHG, S. Diego, October 2018
31. Nolin S, Glicksman A, Tortora N, Allen E, Sherman S, Mila M, Macpherson J, **Vianna-Morgante AM**, Dobkin C, Latham G, Hadd A. AGG Interruptions Lost in Contractions of Maternal Premutation Alleles. 3rd International Conference on FMR1 Premutation, Jerusalem, Israel, September 5-7 2017.
32. Oliveira D, Assoni AF, Carvalho VM, Nishimura AL, Griesi-Oliveira K, Caires LC, Goulart EG, Olavio TR, Alves LM, **Zatz M**. Immunoprecipitation analysis of fused in sarcoma (fus) interacting proteins in neuroprogenitor cells highlights its pathological process in amyotrophic lateral sclerosis. International Congress of ISSCR, Australia, June 2018
33. Passos-Bueno MR, Costa CIS, Montenegro EM; Moreira ES, Costa SS; Lourenço N; **Rosenberg C**, **Krepischi A**; Silva IMW. PgmNr 2421: Cadherins matter to autism spectrum disorders: Which ones? Poster ASHG San Diego 2018
34. Raíces J, Otto P, **Vibranovski MD**. Haploid selection model for new gene, 59th Annual Drosophila Research Conference, Philadelphia, PA, EUA, 2018.
35. Raíces J, Otto P, **Vibranovski MD**. Resolving differences on the chromosomal distributions of Drosophila new genes, Annual Meeting of the Society for Molecular Biology and Evolution (SMBE), Austin, TX, EUA, 2017.
36. Ribeiro Junior AF, Ishiba R, Fernandes SA, Guerrieri DA, Almeida CM, Santos ALF, Souza LS, **Vainzof M**. Muscle satellite cells and impaired late stage regeneration in different murine models of muscular dystrophies. 23rd International congress of the WMS, Mendoza, Argentina, 2-6 october, 201
37. Rivas MP, Aguiar TFM, Maschietto M, Costa C, Toledo SRC, Carraro DM, **Rosenberg C**, **Krepischi A**. Epigenetic mechanisms in liver tumors: gene expression analysis of the epigenetic machinery in hepatoblastomas, SIOP 2017, Washington –USA.
38. Souza L, Almeida C, Silva L, Pavanello R, Gurgel-Gianneti J, Zanoteli E, **Zatz M**, Otto P, **Vainzof M**. Congenital Myopathies (CNM): P. 141High frequency of manifesting carriers in the recessive X-linked myotubular myopathy, Neuromuscular disorders, 28, S71-S72

39. Souza LS, Almeida CF, Silva LGL, Pavanello RCM, Gurgel-Gianneti J, Zanotelli E, **Zatz M**, Otto PA, **Vainzof M**. X-Linked myotubular myopathy: recessive or partly dominant. 23rd International congress of the WMS, Mendoza, Argentina, 2-6 october, 2018
40. Tasaki L, Ishiba R, Ayub-Guerrieri D, Almeida C, Fernandes SA, Ribeiro Jr AF, **Vainzof M.** Osteopontin expression during chronic and acute muscle injury. WMS meeting of the Morld Muscle Society, Saint Malo, 3-7 october, 2017. Neurom. Disord. 27:S193, 2017.
41. Truzzi DR, Coelho FR, Paviani V, Alves SV, **Netto LES**, Augusto O. Bicarbonate increases peroxiredoxin 1 susceptibility to hyperoxidation In: 19th Biennial Meeting of the Society-for-Free-Radical-Research-International (SFRRI), 2018, Lisbon, Portugal. Free Radical Biology and Medicine. 2018. v.120. p.S37 - S37 Referências adicionais : Estados Unidos/Inglês. Meio de divulgação: Vários
42. Turano HG, Gomes F, Domingos RM, Lincopan N, Gales AC, **Netto LES**. Structural characterization of a newly identified pyocin S8, a proteic antibiotic from Pseudomonas aeruginosa with potential therapeutic applications In: SfRMB 25th Annual conference, 2018, Chicago, IL ,EUA.Free Radical Biology Medicine. Elsevier, 2018. v.128 S1. p.s111 - Referências adicionais : Estados Unidos/Inglês. Meio de divulgação: Vários. Home page: [http://https://www.sciencedirect.com/science/article/pii/S0891584918320318]doi.org/10.1016/j.freeradbiomed.2018.10.271

3. Conferences, Symposia, Round Tables, Lectures

1. **Bertola DR-** Mesa redonda: Endocrinologia e Genética: **"**Genética e Baixa estatura, o exemplo da síndrome de Noonan". XXX Congresso Brasileiro de Genética Médica, VII Congresso Brasileiro da SBTEIM e IV Congresso Brasileiro de Enfermagem em Genética e Genômica, Rio de Janeiro, 14 a 18 de maio de 2017.
2. **Bertola DR-** Mesa redonda: Avanços em Cardiogenética: "RASopatias". XXX Congresso Brasileiro de Genética Médica, VII Congresso Brasileiro da SBTEIM e IV Congresso Brasileiro de Enfermagem em Genética e Genômica, Rio de Janeiro, 14 a 18 de maio de 2017.
3. **Bertola DR-** Mesa Redonda: Investigação e Doenças Genéticas no RN: Novas técnicas moleculares. 24º Congresso Brasileiro de Perinatologia, Natal, 25 a 28 de setembro de 2018.
4. **Bertola DR**- Mesa Redonda: Investigação e Doenças Genéticas no RN: O sequenciamento de nova geração na pesquisa: desvendando as bases moleculares de doenças monogênicas. 24º Congresso Brasileiro de Perinatologia, Natal, 25 a 28 de setembro de 2018.
5. **Krepischi AC**- The genetic landscape of neuroblastomas, ICR, São Paulo, 2017. (Simpósio,Lecture)

6. **Mingroni-Netto RC**- Lecture: “Ancestry and Health in quilombo populations of the Ribeira River Valley”, in the Symposium “Tribute to Oswaldo Frota-Pessoa” , Genética 2017 Brazilian-International Congress of Genetics, Águas de Lindóia, SP.
7. **Mingroni-Netto RC**- Lecture: “Genética e Perda Auditiva”, 29 de outubro de 2018, no Programa de Estudos Pós-Graduados em Fonoaudiologia da PUC-São Paulo.
8. **Mingroni-Netto RC** Lecture in Symposium: ‘Aconselhamento Genético multidisciplinar: histórico e conquistas”. Durante o I Simpósio de Aconselhamento Genético Multidisciplinar, no Instituto de Biociências da USP, 16 de junho de 2018.
9. **Okamoto OK**- Invited speaker: . “Qualificação de processos e produtos de terapia celular para uso clinico”. In: “Terapia celular – Transformando a medicina · 1º Fórum Internacional de Terapia Celular Einstein · XXVI Simpósio Internacional de Hemoterapia e Terapia Celular”. April 6th, 2018. São Paulo/ SP, Brazil.
10. **Netto LES**. Ohr : Estrutura e função de uma enzima antioxidante altamente eficiente e seus papéis na interface patógeno-hospedeiro, 2018. (Seminário,Apresentação de Trabalho) Referências adicionais : Brasil/Português; Local: ICB-USP; Cidade: sao paulo; Evento: seminários semanais do Programa de Pós-graduação em Imunologia do ICB-USP; Inst.promotora/financiadora: Programa de Pós-graduação em Imunologia.
11. **Netto LES** .Roles of Cys-based peroxidases in the responses of pathogenic and non pathogenic bacteria, 2018. (Seminário,Apresentação de Trabalho) Referências adicionais : Brasil/Português; Evento: Seminarios Gerais do Departamento de Bioquimica.
12. **Netto LES** Ohr are Cys-based enzymes that reduce fatty acid peroxides and peroxynitrite with extraordinary efficiency and that are not only present in bacteria, but also in the mitochondria of fungi. Possible implications for virulence, 2017. (Congresso,Apresentação de Trabalho) Referências adicionais : Espanha/Inglês. Meio de divulgação: Vários Thiol oxidation in toxicity and signalling 17-21 September 2017 | Sant Feliu de Guixols, Spain; Cidade: Sant Feliu de Guixols, Spain; Evento: Thiol oxidation in toxicity and signalling; Inst.promotora/financiadora: EMBO
13. **Okamoto OK**- Invited speake. “Inovação em terapia celular”. In: EPM: 85 anos de Excelência na Educação Médica - Simpósio Novas fronteiras da Medicina – “Simpósio Novas fronteiras da Medicina – Novartis. August 17th, 2018.
14. **Okamoto OK**- Invited speake: “CRISPR-Cas9 e identificação de alvos terapêuticos em células-tronco tumorais”. In: SIMPÓSIO USP-DISCUTE: Impactos da nova técnica de edição de genomas CRISPR-Cas9 na ciência e na sociedade. May 18th, 2017. São Paulo/ SP, Brazil.
15. **Okamoto OK**- Invited speake: “Stem cell self-renewal genes as drivers of brain tumor aggressiveness”. Scientific meetings of the Biological Sciences Institute at the University of Southampton. March 30th, 2017. Southampton, UK.
16. Palma FR (PhD student), **Netto LES**. Proteostasis Impairment an Endoplasmic Reticulum Stress in a Yeast Model for Amyotrophic Lateral Sclerosis, 2017. (Simpósio,Apresentação de Trabalho) Referências adicionais : República Tcheca/Inglês. . Home page:

http://www.yeast2017.cz/ Flavio Romero Palma (former PhD student) Luis Eduardo Soares Netto (Supervisor) Short talk “Proteostasis Impairment an Endoplasmic Reticulum Stress in a Yeast Model for Amyotrophic Lateral Sclerosis” in the workshop on Proteostasis, Ageing and Disease Models organized by Yury Chernoff at the 28th International Conference on Yeast Genetics and Molecular Biology in Prague. This workshop is scheduled from 5:00-7:00 PM on Thursday August 31, 2017. ; Cidade: Prague; Evento: 28th International Conference on Yeast Genetics and Molecular Biology (ICYGMB); Inst.promotora/financiadora: Yeast Genetics and Molecular Biology.

17. **Rosenberg C**: Challenges of genetic diagnosis in Brazil (Conference at the 5th Central Brazil Cytogenetic Meeting, Goiás, October 17-19 2018).
18. **Vainzof M** – Palestra: “Calpainopathies outside Europe” no “233rd ENMC International Workshop: Clinical Trial readiness for Calpainpathies. 15-17 september, 2017, Naarden, Holand.
19. **Vainzof M** - Palestra: “Diagnóstico Molecular das Miopatias: Estado da Arte”. XXI Fórum de Molestias Neuromusculars da Academia Brasileira de Neurologia. Campinas, 17-18 de agosto de 2018
20. **Vainzof M** - 13º Congresso Brasileiro de Neurologia Infantil. Mesa Redonda: Hot Topics. Palestra: Terapia Gênica: onde estamos? Belo Horizonte, 30 de outubro a 2 de novembro de 2018
21. **Vianna-Morgante AM.** Lecture: “O Titulo de Especialista em Aconselhamento Genético”. - I Simpósio de Aconselhamento Genético Multidisciplinar: Atuação e Perspectivas, Instituto de Biociências, Universidade de São Paulo, São Paulo, SP, 16/06/2018.
22. **Vianna-Morgante AM**. Lecture: Citogenética Humana: Caminhos Trilhados e Perspectivas - I Simpósio de Genética Médica do Centro Oeste – Homenagem aos 30 Anos do Serviço de Genética do HUB/UnB, Brasíla, 7/10/17.
23. **Vianna-Morgante AM**. Lecture: “Oswaldo Frota-Pessoa: A successful three-lane road in science”. In the Symposium - “Tribute to Oswaldo Frota-Pessoa on the centenary of his birth: Following his pathways in science. (Coordinator). GENÉTICA 2017- Brazilian International Congress of Genetics (63º Congresso Brasileiro de Genética), Águas de Lindóia, SP, 13/9/2017.
24. **Vibranovski MD-** Selected Speaker: **Vibranovski MD**, Haploid selection on male germline and the origin of new genes, Germ Cells - Cold Spring Harbor meetings, October, 2018
25. **Vibranovski MD-** Invited Speaker**: Vibranovski MD**, Spermatogenesis expression and evolution of New Genes**,** Third SCLS-CBIS Joint Life Science Research Workshop Evolution of Genes and Genomes, Chengdu, China.
26. **Vibranovski MD -**Invited Speaker: **Vibranovski MD**, Haploid selection and the origin of new genes, Center for Systems Biology in Soochow University, Soochow, China
27. **Vibranovski MD -**Invited Speaker: **Vibranovski MD**, The Use of Genomic and Gene Expression Large-Scale Data for the Analyses of Sexual Evolution, Department of Applied Mathematics, Xi'an Jiaotong University, Xi’an, China.

28. **Vibranovski MD -**Round Table: **Vibranovski MD**, Petrov D. Community, Connections, & Lunch event: Evolutionary & Population Genetics, 59th Annual Drosophila Research Conference, Philadelphia, EUA.
29. **Zatz M.** O futuro da GenÈTICA. I Simpósio de Aconselhamento Genético Multidisciplinar: Atuação e Perspectivas. Instituto de Biociências-USP. 18 de maio de 2018.
30. **Zatz M.** Doenças Neuromusculares: Genes, Aconselhamento Genético. I Congresso Brasileiro de Neurogenética. Academia Brasileira de Neurologia e Associação Paulista de Medicina. São Paulo, 23/24 de março de 2018.
31. **Zatz M.** Parnorama da Terapia Celular no Brasil. 1º Fórum Internacional de Terapia Celular. Albert Einsten. São Paulo, 5 de abril de 2018.
32. **Zarz M.** Palestra "Genética: Escolhas que nossos avós não faziam". Ciclo de Palestras Mulheres e seus Saberes. Fundação Ema Klabin. São Paulo, 28 de abril de 2018.
33. **Zatz M**. Seminário "Segredos do Zica Vírus". Faculdade de Ciências Farmacêuticas da USP. São Paulo, 10 de maio de 2018.
34. Branco S, Guimaraes C, Mostacada J, **Zatz M**. Painel de ciência: Biologia é o novo digital. Festival Prisma da Globonews, 5 de maio de 2018.
35. **Zatz M**. Conferência "Um olhar para a GenÉtica". Academia Nacional de Medicina. Rio de Janeiro, 3 de maio de 2018.
36. Zatz M. Palestra "Células-Tronco: aplicações terapêuticas – Associação Paulista de Medicina. São Paulo, 25 de maio de 2018.
37. **Zatz M**. Conferência "Um olhar para a GenÉtica". Centro de Estudos do Hospital Samaritano. Rio de Janeiro, 3 de maio de 2018.

Annex 3
Theses and Dissertations, Awards

1. Ph.D.

2. **Carolina Malcher Amorim de Carvalho Silva.** Detecção de doenças genéticas fetais através de teste pré-natal não invasivo utilizando sequenciamento de nova geração**.** Instituto de Biociências. USP 9 de Agosto de 2017. Orientador Maria Rita Passos
3. **Lucas Alvizi Cruz**. Título: Genetic and epigenetic mechanisms in the aetiology of orofacial clefts. Biologia/Genética. Instituto de Biociências. USP. 29 de setembro de 2017. Orientador: Maria Rita Passos Bueno.
4. **Natalia Cestari Moreno**. Título: "Efeitos da luz UVA em células de pacientes com Xeroderma Pigmentosum Variante." Biologia/Genética, Instituto de Biociências, USP, 11 de outubro de 2017. Orientador: Mayana Zatz. Atualmente é pós doutoranda ICB, USP.
5. **Tiago Antonio de Souza.** Título: "Análise das alterações genéticas em exomas de camundongos", Programa Interunidades em Biotecnologia, Universidade de São Paulo, SP, em 27 de março de 2018. Orientador: Mayana Zatz. Atualmente é bolsista TT5, ICB, USP.

2. Master

1. **Ana Cristina de Sanctis Giradi**. Título: Transtornos do espectro autista em pacientes com pré-mutação do gene FMR1. Aconselhamento genético e Genômica Humana. 5 de marlo de 2018. Orientador: Maria Rita Passos Bueno.
2. **Antonio Fernando Ribeiro Junior**. Título: Potencial de regeneração da relaxina no músculo distrófico. Programa: Biologia-Genética.. 23 de março de 2018 Bolsa CAPES. Orientador: Mariz Vainzof -
3. **Carolina de Seixas Couto Leite**. Título: Envolvimento da proteína SAM68 na regulação da proliferação celular em tumores de sistema nervoso central. 19 de março de 2018. Genética. Ciências Biológicas - Universidade de São Paulo. Orientador: Oswaldo Keith Okamoto.
4. **Cláudia Ismania Simogy Costa**. Título: Copy number variations (CNVs) in Brazilian patients with autism spectrum disorder (ASD). . Biologia/Genética. Instituto de Biociências. Biologia/Genética. Instituto de Biociências . 18 de julho de 2018. Orientador: Maria Rita Passos Bueno.
5. **Rodrigo Salazar Silva** Título: "Análise de expressão de gene candidato à surdez em modelos animais", 9 de novembro de 2017. Ciências Biológicas (Biologia-Genética) - Universidade de São Paulo. Orientador: Regina Célia Mingroni Netto
6. **Rogério Luis Aleixo Silva.** Título: Caracterização estrutural e bioquí¬mica de LsfA, uma 1-Cys Prx envolvida na virulência de Pseudomonas aeruginosa. Ciências Biológicas (Biologia-Genética – USP. 30 de maio de 2018. Orientador: Luis Eduardo Soares Netto..

7. **Stephanie de Alcantara Fernandes**. Título: Comparação e caracterização de células-tronco mesenquimais de medula óssea, tecido adiposo e mioblastos em modelos murinos para distrofias musculares. Programa: Biologia-Genética fev/2015- 4 de Agosto de 2017. Orientador: Mariz Vainzof .- Bolsa FAPESP

3. Awards

1. Premio de melhor trabalho (1º lugar), da Academia Brasileira de Neurologia. Trabalho: A novel complex neurological phenotype due to a homozygous mutation in FDX2 confirmed by molecular and functional studies. Gurgel-Giannetti J. Lynch DS, Paiva ARB, Lucato LT, Yamamoto G, thomsen C, Basu S, Reua F, Giannetti AV, Hirano M, Van Der Knaap MS, Lill R, Vainzof M, Oldfors A, Houlden H, KOk F. Brain. 2018 Jul 13. doi: 10.1093/brain/awy172
2. Premio Elsevier no International Congresso f the World Muscle society. Antonio Fernando Ribeiro Jr. Mendoza, Argentina,
3. Premio Elsevier no International Congresso f the World Muscle society. Lucas Santos e Souza. Mendoza, Argentina.
4. Prêmio Jovem Geneticista 2018**.** Clarissa Ribeiro Reily Rocha, "Mecanismos de resistência a quimioterápicos em células tumorais"., International Congress of Genetics, Foz do Iguaçu, 10-14/09/2018.
5. Premio Biochemical Society - Melhor Poster - aluno Renato Mateus Domingos - orientador Luis Netto, SBBq.
6. 2017 Travel award - aluna de doutorado Anita Del Guercio, Society Redox Biology Medicine

Annex 4

Tables Education /Out Reach

Annex 4.1

Laboratory Class Project - Training of 54 High School teachers

Educational Directory of Center-West Region, February 27th, 2018.

Educational Directory of Osasco Region, February 28 th, 2018.

High School	Teacher	Educational Directory
EE Alcyr de Oliveira Porciuncula	Antonio Pedro de Castro	Osasco
EE Prof. Alice Velho Teixeira	Elen Gonçalves dos Santos	Osasco
EE Dr. Américo Marco Antonio	Sueli G. de Souza Bento	Osasco
EE Antonio Carlos da Trindade	Ingrid dos Santos Ricardo	Osasco
E E Prof. Armando Gaban	Marina Santos Barbosa	Osasco
EE Dr. Aureliano Leite	Enides Barroso	Osasco
EE Prof. Benedito Caldeira	Isabel Cristina Pereira	Osasco
EE Prof. Eloi Lacerda	Andrea Barbieri Rezende	Osasco
EE Prof. Ernesto Thenn de Barros	Maria de Lourdes Mesquita de Oliveira Sepriano	Osasco
EE Prof. Fanny Monzoni Santos	Tânia da Silva Nascimento Cardim	Osasco
EE Prof. Gastão Ramos	Josilaine Ribeiro de Barros	Osasco
EE Graciliano Ramos	Carlos Alberto Ramos	Osasco
EE Dep. Guilherme de Oliveira Gomes	Gislene Mariano Costa Santos	Osasco
EE Prof. Heloisa Assumpção	Sergio Seixas Barros	Osasco
EE Neusa de Oliveira Prévide	Ligia Fernandes de Jesus Bastos	Osasco
EE Prof. Ernesto Thenn de Barros	Marcos Viana da Silva	Osasco
EE Prof. João Batista de Brito	Ezilda Oliveira	Osasco
EE Prof. José Edson Martins Gomes	Mirian Alves Aversa	Osasco
EE José Geraldo Vieira	Valquíria Fornarolli	Osasco
EE Prof. José Jorge	Renato Policarpo da Silva	Osasco
EE Prof. José Liberatti	Lucilene C. Souza	Osasco
EE Prof. José Maria Rodrigues Leite	Cristina Della Matta	Osasco
EE José Ribeiro de Souza	Carolina de Azevedo Stefano Sonia R S Monteiro	Osasco

EE Josué Benedicto Mendes	Maira de Figueiredo Nunes	Osasco
EE Leonardo Villas Boas	Maria Angela da Silva Gomes	Osasco
EE Prof. Lucy Anna Latorre	Marcos Viana da Silva	Osasco
EE Prof. Dr. Luiz Lustosa da Silva	Angela Maria Zanin	Osasco
EE Prof. Maria Augusta Siqueira	Luciana Cardoso Romeiko	Osasco
EE Prof. Neusa de Oliveira Prévide	Alessandra Paula de Andrade	Osasco
EE Prof. Newton do Espirito Santo Ayres	Elias Tavares	Osasco
EE Prof. Orlando Geríbola	Elaine Dias dos Santos	Osasco
Educador Paulo Freire	Carolina de Azevedo Stefano	Osasco
EE Emiliano Augusto Cavalcanti de A e Melo	Jose Alves Mendes Filho	Center-West
EE João XXIII	Cecília Elisabete Batista	Center-West
	Luciene Chaves	Center-West
EE Odair Martiniano da Silva – Mandela	Rosana Laranjeira dos Santos	Center-West
	Raquel Martins	Center-West
EE Pedro Fonseca	Patricia da Silveira Pires	Center-West
EE Pereira Barreto	Ana Claudia da Cunha Mattos	Center-West
EE Prof. Almeida Junior	Sheila Pereira Queiroz Morgadouro	Center-West
EE Ana Rosa de Araujo	Eunice Alves Pereira	Center-West
EE Prof. Architiclino Santos	Samanta Urbach	Center-West
	Jéssica B. N. de Almeida	Center-West
EE Prof. Emygdio de Barros	Larissa Caroline M. Vieira	Center-West
EE Fernão Dias Paes	Lilian Colombini Etchebehere	Center-West
EE Godofredo Furtado	Eliana Oliveira Pescuma	Center-West
EE Profa. José Monteiro Boanova	Silmara Regina Siqueira	Center-West
EE Keizo Ishihara	Gisele Fernandes Agripino	Center-West
EE Prof. Lourival Gomes Machado	Maria Luiza Aldrighi	Center-West
EE Prof. Manuel Ciridião Buarque*	Cecília Vaz Castro	Center-West
	Thatiana O Balduweiz*	Center-West
EE Prof.ª Lygia de Azevedo Souza e Sá*	Ieda Martins Santiago	Center-West
EE Profa. Maria Eugênia Martins	Antenor J. S. de Morais	Centro Oeste
	Giane Ventura Rabelo	Centro Oeste
	Cristiane Palmeira	Centro Oeste

Teaches at 2 different High Schools

Annex 4.2

Training of 58 High School students to act as monitors at their schools.

Laboratory Class Project, Osasco Educational Directory,

February 27th and 28th, 2018.

High School	Student	Educational Directory
EE Prof. Alcyr de Oliveira Porciúncula	Thalita Alexandre Teixeira Joao Victor de Assis M. Miranda Anna Julia M. V. Azevedo	Osasco
EE Prof. Alice Velho Teixeira	Bianca dos Santos Sena	Osasco
EE Cel. Antonio Paiva de Sampaio	Paulo Miguel do Nascimento Evelyn Cavalheiro da Silva	Osasco
E E Prof. Armando Gaban	Isabella Maria dos Santos Soares Jhenipher Souza Leite Luiz Carlos Zennan de Morais	Osasco
EE Prof. Benedito Caldeira	Giovana K. Soares Heron Natã G. Carmélio Gustavo M. Campos	Osasco
EE Prof. Eloi Lacerda	Gabriel Gonçalves de Souza Kessia Santana Lopes Caique F. Soares da Silva	Osasco
EE Prof. Ernesto Thenn de Barros	Nicoly Siqueira Abade Aymei Victória Geovana da Silva Torres	Osasco
EE Graciliano Ramos	Leonizia Nogueira dos Santos Danilo Queiroz Miranda Moura Leticia Vitória de A. Lourenço	Osasco
EE Dep. Guilherme de Oliveira Gomes	Thalia de Oliveira	Osasco
EE Prof. João Batista de Brito	Mel Clemente Bulhoes Isabelli Lima Felix Sophia Regina Ribeiro Delelli	Osasco
EE Prof. Jose Edson Martins Gomes	Giovana Oliveira RR Hagata Vitória Miranda Luiz	Osasco
EE Jose Geraldo Vieira	Ketlyn Cristina C da Silva Caroline Thayane da Silva Luiz Gustavo P. da Silva	Osasco
EE Prof. José Jorge	Brenda Souza Fornarolli Samuel Queiroz Santos	Osasco
EE Prof. José Liberatti	Laura Mayumi Camila Eduarda	Osasco

EE José Maria Rodrigues Leite	Roberth dos S. Bispo Isadora Isaac Ferreira	Osasco
EE José Ribeiro de Souza	Beatriz Simoes de Oliveira Pedro de Oliveira Santos	Osasco
EE Julia Lopes de Almeida	Julia Rutizat Nascimento Raissa Andrade do Nascimento Matheus Alves Landini	Osasco
EE Leonardo Vilas Boas	Luiz Felipe S. de Oliveira Gabriel Vinicius Pereira dos Santos Ketlem Amanda de Oliveira	Osasco
EE Maria Augusta Siqueira	Giovana Gonçalves Lemos Raul Alves do Nascimento Paulo H. Ferreira	Osasco
EE Neusa de Oliveira Prévide	Mycaele Vitoria F. da Silva Juliana Albuquerque Soares Amalia Alves Amorim	Osasco
EE Oguiomar Ruggieri	Gizelly Queiroz Lacerda Gabriel Felipe O. Ney Laura Carolina Daniel Oliveira Ribeiro Lucas Dias Carneiro	Osasco
EE Prof. Orlando Geríbola	Rafaella Santos da Silva Laysa Gomes do Nascimento	Osasco

Annex 4.3

Laboratory at School Project – 94 Schools were attended

from July, 2017 to June, 2018

*Schools attended twice in this period

High School	Educational Directory
EE Dona Ana Rosa de Araújo*	Center-West
EE Senador Adolfo Gordo	Center-West
EE Lucy Anna Latorre*	Osasco
EE Armando Gaban*	Osasco
EE José Liberatti*	Osasco
EE Coronel Antonio Paiva Sampaio	Osasco
EE Martim Francisco	Center-West
EE Prof. Alberto Levy	Center-West

EE Gloria Azedia Bonetti*	Osasco
EE Graciliano Ramos*	Osasco
EE Américo Marco Antônio*	Osasco
EE Claudinei Garcia*	Osasco
EE Romeu de Moraes*	Center-West
EE Prof. Manuel Ciridião Buarque*	Center-West
EE Rosa Bonfiglioli*	Osasco
EE Maria Augusta Siqueira*	Osasco
EE Antonio Raposo Tavares*	Osasco
EE Aureliano Leite*	Osasco
EE Anhanguera	Center-West
EE Pereira Barreto*	Center-West
EE Ricardo Genésio da Silva	Osasco
EE Alcyr Porciúncula	Osasco
EE Fanny Manzoni Santos*	Osasco
EE Newton Espírito Sabato Ayres*	Osasco
EE Prof. Lourival Gomes Machado*	Center-West
EE Odair Martiniano da Silva - Mandela	Center-West
EE José Maria Rodrigues Leite*	Osasco
EE José Geraldo Vieira*	Osasco
EE Irmã Gabriela*	Osasco
EE Paulo Freire*	Osasco
Fundação Casa 1	Osasco
Fundação Casa 2	Osasco
EE Maria Augusta Siqueira	Osasco
EE Heloísa Assumpção	Osasco
EE Alice Velho Teixeira	Osasco
EE Major Telmo	Osasco
EE Cel Antonio Paiva	Osasco
EE José Jorge	Osasco
EE Aureliano Leite	Osasco
EE Francisca Lisboa Peralta	Osasco
EE José Edson	Osasco
EE Fernando Buonaduce	Osasco
EE Neuza de O. Prévide	Osasco
EE Deputado Guilherme O. Gomes	Osasco
EE Ernesto Thenn de Barros	Osasco
EE José Ribeiro de Souza	Osasco

EE Orlando Geríbola EE	Osasco
EE Oguiomar Ruggeri	Osasco
EE Benedito Caldeira	Osasco
EE Josué Benedicto Mendes	Osasco
EE Luis Lustosa Filho	Osasco
EE Eloi Lacerda	Osasco
EE Leonardo Vilas Boas	Osasco
EE Antônio Carlos da Trindade	Osasco
EE Tarsila do Amaral	Osasco
EE Gastão Ramos	Osasco
EE João XXII	Center-West
EE Solon Borges dos Reis	Center-West
EE Prof Architiclino Santos	Center-West
EE Napoleão de Carvalho Freire	Center-West
EE Fernão Dias Paes	Center-West
EE Prof. Almeida Júnior	Center-West
EE Maria Eugênia Martins	Center-West
EE Profa.Lygia de Azevedo Souza e Sá	Center-West
EE Prof. Pedro Fonseca	Center-West
EE Godofredo Furtado	Center-West
EE Virgília Rodrigues A. de Carvalho Pinto	Center-West
EE Prof. José Monteiro Boanova	Center-West
EE Prof. Oswaldo Walder	Center-West
EE Prof. Emygdio de Barros	Center-West
EE Odair Martiniano da Silva - Mandela	Center-West

Annex 4.4

Training of 44 High School teachers to participated at the Instructional Support Project.

May 15th, 2018

High School	Teacher	Educational Directory
EE Prof. Alice Velho Teixeira	Elen Gonçalves dos Santos	Osasco
EE Dr. Américo Marco Antonio	Beatriz Ribeiro Zanon	Osasco
EE Antônio de Almeida Junior	Izilda Aparecida da Silva	Osasco

EE Cel. Antônio Paiva Sampaio	Júlio Eduardo R Nogueira	Osasco
EE Antônio Raposo Tavares	Thiago de Oliveira Nogueira	Osasco
E E Prof. Armando Gaban	Marina Santos Barbosa	Osasco
EE Dr. Aureliano Leite	Jennifer Caroline de Sousa	Osasco
EE Prof. Benedito Caldeira	Isabel Cristina Pereira	Osasco
EE Prof. Eloi Lacerda	Andrea Barbieri Rezende	Osasco
EE Prof. Ernesto Thenn de Barros	Marcos Viana da Silva*	Osasco
EE Prof. Fernando Buonaduce	Eneida Domingues Fernandes	Osasco
EE Prof. Francisca Lisboa Peralta	Roseli Cristina Laranjeira	Osasco
EE Prof. Francisco Casabona	Carolina Assaf Iseri	Osasco
EE Francisca Matarazzo Sobrinho	Karina Almeida	Osasco
EE Prof. Gastão Ramos	Alessandra Brito Santos de Freitas	Osasco
EE Graciliano Ramos	Carlos Alberto Ramos	Osasco
EE Dep. Guilherme de Oliveira Gomes	José Marton Beraldi	Osasco
EE Prof. Heloisa Assumpção	Carolina Assaf Iseri*	Osasco
Jardim Santa Maria III	Alline Ramos Pereira do Nascimento	Osasco
EE Prof. João Baptista de Brito	Ezilda O. Alves	Osasco
EE José Edson Martins Gomes	Mirian Alves Aversa	Osasco
EE José Geraldo Vieira	Valquíria Fornarolli da Cruz	Osasco
EE Prof. José Jorge	Renato Policarpo da Silva	Osasco
EE Prof. José Liberatti	Lucilene Costa de Souza	Osasco
EE Prof. José Maria Rodrigues Leite	Cristina Della Matta	Osasco
EE José Ribeiro de Souza	Sônia R. S. Monteiro	Osasco
EE Prof. Josué Benedicto Mendes	Maíra Figueiredo Nunes	Osasco
EE Julia Lopes Almeida	Amanda Gouveia da Silva	Osasco
EE Leonardo Villas Boas	Maria Angela da S. Gomes	Osasco
	Grazielle Macedo de França Costa	Osasco
EE Prof. Lucy Anna Latorre	Marcos Viana da Silva	Osasco
EE Prof. Dr. Luiz Lustosa da Silva	Suellen Ribeiro Borde	Osasco
EE Prof. Maria Augusta Siqueira	Luciana Cardoso Romeiko	Osasco
EE Major Telmo Coelho Filho	Cristiane de M. Potença Cavalli	Osasco

EE Prof. Neusa de oliveira Prévide	Guilherme T. B. Mazzini	Osasco
	Viviane dos Reis Silva	Osasco
EE Prof. Newton do Espírito Santo Ayres	Elias Tavares	Osasco
EE Prof. Oguiomar Ruggieri	Natalia Rosa Sciani	Osasco
Fundação Casa I Fundação Casa II	Miriam Santana S. Aparecida	Osasco
EE Prof. Orlando Geríbola	Rubileide Santos	Osasco
EE Educador Paulo Freire	Carolina de Azevedo Stefano	Osasco
EE São Paulo da Cruz	Renato Policarpo da Silva	Osasco
EE Tarsila do Amaral	Roberto Masaru Ueda	Osasco
EE Prof. Vicente Peixoto	Luiz Alfredo Favaretto	Osasco

Annex 4.5 (a and b)

(a) Training of 27 High School teachers of Biology and Science to participate at the Project Scientific Exhibitions

(b) (Light and Life + Giant Cell).

Barretos (State of São Paulo), September 19th, 2017.

High School	Teacher	Educational Directory
EE Alice Fontoura de Araujo	Ricardo de Jesus Ribeiro Faleiros	Barretos
EE Cel Almeida Pinto	Walter Hermogenes da Costa Filho	Barretos
EE Dona Anita Costa	Cesar Rodrigues Castanheira	Barretos
E E Dr. Antonio A. Reis Neves	Ivanice Cristina Peres da Silva	Barretos
EE Dr. Antonio Olympio	Erika Akiko Kaneda	Barretos
EE Prof. Benedito P. Cardoso	Eliane Aparecida Monteiro	Barretos
CEEJA	Derik Mateus Martoneto	Barretos
EE Dalva Lellis G. Prado	Paulo Rogério Lago	Barretos
EE Prof. Dalva Vieira Itavo	José Roberto Zata	Barretos
EE Prof. Darcy Silveira Vaz	Edna Lopes Montegro Luciana Cunha Sabino	Barretos
EE Dona Anita Costa	Cesar Rodrigues Castanheira	Barretos
EE Dr. Eloi Lopes Ferraz	Mileni Delgado	Barretos
EE Enoch Garcia Leal	Vandeir de Moura Gonçalves	Barretos
EE Fábio Junqueira Franco	João Daushas Junior	Barretos
EE José Antonio Santana	Sebastião Rui Martins	Barretos

EE José Marcelino de Almeida	Érica Cristiane Basso Pignatari	Barretos
EE Prof. Lacy B. de Souza	Maria Cristina Senhorini Silva	Barretos
EE Embaixador Macedo Soares	Rildo Vasconcelos	Barretos
EE Prof. Maria Helena Scannavino	Rodrigo Branco Gonçalves	Barretos
EE Prof. Maria U.B. Furquim	Priscila Moreda Perrone	Barretos
E E Mario Vieira Marcondes	Dorival Aparecido da Silva	Barretos
EE Ovidio S. Dias	Juliana Gonçalves	Barretos
EE Prof. Paulina N. Moraes	Marcos Henrique Aparecido da Costa	Barretos
EE Cel. Silvestre de Lima	José Antonio Rodrigues de Souza	Barretos
EE Dr. Wilquem Manoel Neves	Ana Lucia Castro Pimenta Souza	Barretos
EE Zezinho Portugal	Adriano da Silva Pereira	Barretos

(c) Training of 44 High School teachers of Physics to participate at the Project Scientific Exhibitions (Light and Life + Giant Cell).

Barretos (State of São Paulo), September 19th, 2017.

High School	Teacher	Educational Directory
EE Dona Anita Costa	Ricardo Soares Pimenta	Barretos
E E Dr. Antonio A. Reis Neves	João Paulo Lins de Lima	Barretos
EE Dr. Antonio Olympio	Paulo Ribeiro Rosa	Barretos
EE Prof. Benedito P. Cardoso	Magda Ane dos Santos Ferreira	Barretos
EE Prof. Aymore do Brasil	Sueli Scofoni da Costa Vilela	Barretos
CEEJA	Marcela Alves do Nascimento	Barretos
EE Prof. Dalva Vieira Itavo	Lilian Cristina Bordilhão	Barretos
EE Prof. Darcy Silveira Vaz	Luciana Cunha Sabino	Barretos
EE Dr. Eloi Lopes Ferraz	Ana Marcia do Nascimento Bigui	Barretos
EE Enoch Garcia Leal	Rosilene Correa Ramos	Barretos
EE Com. Francisco B. Ferreira	Rosana Silva	Barretos
EE José Antonio Santana	Adriana Silva Matos Aguiar	Barretos
EE José Marcelino de Almeida	Ana Paula Campana Furlanetto Lopes	Barretos
EE Prof. Lacy B. de Souza	Solange Cruz de Barcellos São Romão	Barretos
EE Embaixador Macedo Soares	Aparecida Pereira Rildo Vasconcelos	Barretos
EE Prof. Maria Helena	Edilene Lopes de Souza	Barretos

Scannavino		
EE Prof. Maria U.B. Furquim	João Paulo Olmos de Souza	Barretos
E E Mario Vieira Marcondes	Valéria Ribeiro Moura	Barretos
EE Cap. Narciso Bertolino	Luciana de Cássia Mendes	Barretos
EE Prof. Paulina N. Moraes	Flávia Regina De Lima	Barretos
EE Cel. Silvestre de Lima	Elisangela Quintanilha	Barretos
EE Valois Scortecci	Leonice Terezinha Stefanini	Barretos
EE Dr. Wilquem Manoel Neves	Cristiane Silva Vilarinho	Barretos

Annex 4.5

Exbitions of the "Giant Cell" and the Scientific Exhbition "Light and Life"

Giant Cell

- August, 23 to 26, 2017 - "USP e as Profissões", Parque Cientec, 2800 visitors. Reportagem Globo - http://g1.globo.com/sao-paulo/sptv-2edicao/videos/t/integras/v/sp2-edicao-de-quinta-feira-24082017/6102035/
- August, 18 to 20, 2018 – "USP e as Profissões" - Parque Cientec. 2,500 visitors.

Giant Cell and Light and Life

- September, 18 to 22, 2017 - EE Profa. Maria Helena Scanavinno, Barretos, SP, 1.500 visitors
- April, 09 to 13, 2018 – Serramar Parque Shopping, 900 visitors32 estudantes de 21 escolas, 37 professores, 215 pessoas público espontâneo, 16 funcionários Shopping – Total 900 pessoas

Annex 4.6

Interviews to the Media and Science Dissemination Articles

2017

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Annex 5
Personnel

Students IC

Name	Supervisor
Alice Kei Endo	Oswaldo Keith Okamoto
Annie Tomoe Takaesu	Ana C. V. Krepischi
Camila Avila Martins	Regina C. Mingroni-Netto
Artur Berselle	Maria Rita Passos-Bueno
Barbara Santos de Oliveira	Merari F. R. Ferrari
Beatriz Dias Barbieri	Oswaldo Keith Okamoto
Camila Corradi	Carlos F M Menck
Camila Lovaglio Santos	Maria Rita Passos-Bueno
Carolina de Seixas Couto Leite	Oswaldo Keith Okamoto
Dione Oliveira Jordan	Oswaldo Keith Okamoto
Eduardo Tsuchida	Luis Eduardo Soares Netto
Erika Ramos	Maria Rita Passos-Bueno
Gabriel Monteiro do Carmo	Mayana Zatz
Gabriela Furukawa	Oswaldo Keith Okamoto
Gabriele da Silva Campos	Maria Rita Passos-Bueno
Gabriella Hsiya	Maria Rita Passos-Bueno
Joana Guiro Carvalho da Rocha	Oswaldo Keith Okamoto
João Vicente Malvezzi	Ana C. V. Krepischi
Karla Pacheco Melo	Merari F. R. Ferrari
Kayque Alves Telles Silva	Mayana Zatz
Letícia Yumi Takasi	Mariz Vainzof
Leonardo da Costa Carvalheira	Oswaldo Keith Okamoto
Lucas Vecchiato de Melo	Maria Rita Passos-Bueno
Maíra Fessardi	Luis Eduardo Soares Netto
Matheus Molina	Carlos F M Menck
Natália Fagundes Borges Teruel	Merari F. R. Ferrari
Niara Régia F. de Souza	Angela M. Vianna-Morgante
Rafaela Regina Cardoso	Merari F. R. Ferrari
Rosanna Miki Kimura Cerioni	Oswaldo Keith Okamoto
Taiany Curdulino	Ana C. V. Krepischi
Thais Alexandre Falkembach Andreis	Merari F. R. Ferrari
Thiago Giove	Oswaldo Keith Okamoto
Vitória Alves Lima	Oswaldo Keith Okamoto

Students Master

Name	Supervisor
Amanda Faria Assoni	Oswaldo Keith Okamoto
Aline Lopes Ribeiro	Oswaldo Keith Okamoto
Alex Marcel Moreira Dias	Regina C.Mingroni-Netto
André Silva Bueno	Regina C.Mingroni-Netto
Caissa Calazans	Carla Rosenberg
Camila Correia Avelino	Maria Dulcetti Vibranovski
Carolina de Athayde Mendonça	Maria Dulcetti Vibranovski
Debora Camilotti	Carla Rosenberg
Francine Campagnari	Carla Rosenberg
Frederico Monfardini	Maria Dulcetti Vibranovski
Gabriela Novaes	Oswaldo Keith Okamoto
Giovanna Tolezano	Ana C. V. Krepischi
Jaina Araújo Reia	Merari F. R. Ferrari
Juliana Plat de Aguiar Gomes	Mayana Zatz
Juliana Sobral de Barros	Ana C. V. Krepischi
Larissa Antunes Nascimento	Regina C.Mingroni-Netto
Letícia Alves da Rocha	Debora R. Bertola
Leonardo Galleni Leão da SIlva	Mariz Vainzof
Livia Luz Souza Nascimento	Frederico Menck
Lucas Santos e Souza	Mariz Vainzof
Mariana Soares Fogo	Celia Koiffmann
Mara Dell'Ospedale Ribeiro	Fernando Kok
Mayra Pelatti	Mayana Zatz
Raquel de Souza Lima	Merari F. R. Ferrari
Ricardo di Lazzaro Filho	Debora R. Bertola
Rogério Luis Aleixo Silva	Luis Eduardo Netto
Sandra Mabel Sánchez Sánchez	Andrea Sertie
Vanessa Simões	Luis Eduardo Netto
William Bertani Torre	Regina C.Mingroni-Netto

Students Doctorate

Name	Supervisor
Ágatha Cristhina Oliveira Faria	Maria Rita Passos-Bueno
Allysson Allan De Farias	Fernando Kok
Amanda Faria Assoni	Mayana Zatz
Angela May Suzuki	Maria Rita Passos Bueno
Anita Martins Fontes Del Guercio	Luis Eduardo Soares Netto

Camila de Freitas Almeida	Mariz Vainzof
Camila Manso Musso	Maria Rita Passos Bueno
Carolina de Oliveira Rodini	Oswaldo Keith Okamoto
Carolina Malcher	Maria Rita Passos-Bueno
Carolini Kaid Dávila	Oswaldo Keith Okamoto
Danielle de Paula Moreira	Maria Rita Passos-Bueno
Danyllo Felipe de Oliveira	Mayana Zatz
Davi Mendes	Carlos F M Menck
Davi Jardim Martins	Carlos F M Menck
Eduarda Morgana da Silva M. M. de Souza	Maria Rita Passos Bueno
Ernesto Goulart	Mayana Zatz
Estela Mitie Cruvinel	Célia P. Koiffmann
Felipe Augusto André Ishiy	Maria Rita Passos-Bueno
Fernando Gomes	Luis Eduardo Soares Netto
Francine Campagnari	Carla Rosenberg
Gabriel Nassar Reich Goldstein	Maria D. Vibranovski
Gustavo Satoru Kajitani	Carlos F M Menck
Juliana Emilia Prior Carnavalli	Carla Rosenberg
Laura Carvalho	Carla Rosenberg
Luiz Gustavo Dufner De Almeida	Luciana Haddad
Maria Prates Rivas	Ana C. V. Krepischi
Michelle Buscarilli de Moraes	Debora Romeo Bertola
Nathalia Quinteros	Carlos F M Menck
Patrícia Benites Gonçalves da Silva	Oswaldo Keith Okamoto
Renata Bannitz Fernandes	Luis Eduardo Soares Netto
Renato Domingos	Luis Eduardo Soares Netto
Renato Mateus Domingos	Luis Eduardo Soares Netto
Rodrigo Atique Ferraz de Toledo	Maria Rita Passos Bueno
Silvia Costa	Carla Rosenberg
Talita Aguiar	Ana C. V. Krepischi
Tatiana Ferreira de Almeida	Maria Rita Passos-Bueno
Thaiany Quevedo Melo	Merari F. R. Ferrari
Tiago Antonio de Souza	Carlos F M Menck
Vinícius Magalhães Borges	Regina C. Mingroni-Netto

Students Pos Doctorate /Visiting Researcher

Name	Supervisor
Alexandra Pelegrini	Carlos F. M. Menck
Amajad Iqbal Kazi	Merari F. R. Ferrari
André Luis Fernandes dos Santos	Mariz Vainzof

Anne Caroline Barbosa	Ana C V Krepischi
Beatriz de Araujo Cortez	Oswaldo Keith Okamoto
Bruno Henrique Silva Araujo Torres	Esper Abrão Cavalheiro
Carla Sustek D'Angelo	Célia P. Koiffmann
Clarissa R. Ribeiro Rocha	Carlos F. M. Menck
Danielle Moreira	Maria Rita Passos-Bueno
Darine Villela	Carla Rosenberg
Diogo de Abreu Meireles	Luis Eduardo Soares Netto
Eder Zucconi	Mayana Zatz
Gerson Shigeru Kobayashi	Maria Rita Passos-Bueno
Giovana Leandro da Silva	Carlos F. M. Menck
Giuliana Castello Coatti	Mayana Zatz
Karina Griesi Oliveira	Maria Rita Passos-Bueno
Ligia Pereira de Castro	Carlos F. M. Menck
Lucas Alvizi	Maria Rita Passos-Bueno
Lúcia Inês Macedo de Souza	Mayana Zatz
Luciana Rodrigues Gomes	Carlos F. M. Menck
Luciano Abreu Brito	Maria Rita Passos-Bueno
Luiz Carlos de Caires Júnior	Mayana Zatz
Márcia Cristina Teixeira dos Santos	Oswaldo Keith Okamoto
Maria Cristina Mingues Spinola	Luis Eduardo Soares Netto
Mariane Secco	Mayana Zatz
Natale Cavaçana	Mayana Zatz
Roberto Dalto Fanganillo	Maria Rita Passos-Bueno
Uirá Souto Melo	Mayana Zatz
Valquíria Santos	Carlos F. M. Menck
Vivek Kumar	Oswaldo Keith Okamoto

Laboratory Technicians and Assistants

Name	Funding Source	Supervisor
Andressa Yurie Silvestre Sakugawa	USP	Merari R. F. Ferrari
Cláudia Irene Emilio de Castro Fabris	USP	Celia Koiffmann
Daiane Gil Franco	INCT/FUSP	Maria Rita Passos-Bueno
Gilberto Stam	CEGH-CELL/ FUSP	Eliana M.B. Dessen
Guilherme Lopes Yamamoto	CEGH-CELL/ FUSP	Maria Rita Passos-Bueno
Heloísa Maria de Siqueira Bueno	CEGH-CELL/ FUSP	Mayana Zatz
Jaqueline Yu Ting Wang	INCT	Maria Rita Passos-Bueno
Job Carvalho Bezerra	USP	Eliana M.B. Dessen
Kátia Maria da Rocha	CEPID / USP	Maria Rita Passos-Bueno

Kelly Bagatini	CEGH-CELL/ FUSP	Maria Rita Passos-Bueno
Letícia Nogueira Feitosa	CEGH-CELL/ FUSP	Mariz Vainzof
Maria Raimunda L. Santana Pinheiro	USP	Ana C. V. Krepischi
Marília de Oliveira Scliar	INCT/FUSP	Maria Rita Passos-Bueno
Marta Canovas	CEPID / USP	Mayana Zatz
Meire Aguena	CEGH-CELL/ FUSP	Maria Rita Passos-Bueno
Monica Castro V. Rodrigues da Silva	CEGH-CELL/ FUSP	Maria Rita Passos-Bueno
Monize Lazar Magalhães	CEPID / USP	Maria Rita Passos-Bueno
Naila Cristina V. Lourenço	INCT / USP	Maria Rita Passos-Bueno
Patricia Semedo Kuriki	INCT / USP	Oswaldo Keith Okamoto
Paulo Rogério de Souza	USP	Regina C.Mingroni-Netto
Roberto Rivelino de Camargo	CEPID / USP	Maria Rita Passos-Bueno
Silvia Costa	USP	Carla Rosenberg
Simone Gomes Ferreira	USP	Maria Rita Passos-Bueno
Simone Vidigal Alves	USP	Luis Eduardo Soares Netto
Thais Oliveira de Andrade	AACD/ FUSP	Mayana Zatz
Tatiane Viana	CEGH-CELL/ FUSP	Maria Rita Passos-Bueno
Thiago Geronimo Alegria	USP	Luis Eduardo Soares Netto
Vanessa Naomi V. O. Takahashi	CEPID / USP	Maria Rita Passos-Bueno
Vivian Landini e Silva	AACD/ FUSP	Mayana Zatz

Bioinformatics Support / Information Technology

Name	Funding Source	Supervisor
Carlos Eduardo da Silva Simões	INCT/FUSP	Maria Rita Passos-Bueno
Daniel Bozoklian do Amaral	INCT	Maria Rita Passos-Bueno
Diego Lima de Carvalho	INCT	Maria Rita Passos-Bueno
Ricardo Nonaka	USP	Maria Rita Passos-Bueno

Administrative

Name	Funding Source	Supervisor
Ana Carolina de Moura Ferreira	CEGH-CELL/ FFM	Maria Rita Passos-Bueno
Constancia Urbani Gotto	AACD/FUSP	Mayana Zatz
Luceleni da Silva	USP	Celia P. Koiffmann
Luciana Cristina A.Oliveira	CEPID / USP	Eliana M.B. Dessen
Luciano Cabral da Silva Costa	USP	Mariz Vainzof
Maraisa de Castro Sebastião	USP	Ana C. V. Krepischi
Márcia Góes Teixeira	AACD/ FUSP	Mayana Zatz
Marta Rita Celestino de Macedo	CEPID / USP	Mayana Zatz
Wagner Falciano	CEPID / USP	Mayana Zatz