Our mission is to support and fund basic science or clinical research for childhood brain tumors. We are dedicated to heightening public awareness of this devastating disease, and improving the quality of life for those that it affects by funding vital research initiatives.

This year’s gala was held at Madame Tussaud’s in Washington DC on November 10, 2018, chaired by Dr. Roger and Bashi Packer. The event was a huge success with a record turnout. A dedicated committee formed by the Packer’s came together to plan an exciting evening that focused on recognizing CBTF and the accomplishments of several individuals and groups over the past 25 years. Bashi Packer thanked everyone for attending and thanked all of the volunteers, committee members, and Joyce Kammerman for the time commitment given to the event. She graciously acknowledged all of the sponsors, supporters, and friends for sponsoring and attending the anniversary event.

Throughout the evening attendees enjoyed posing with friends and colleagues with the celebrities and political figures. Photos will be posted on the CBTF website: www.childhoodbraintumor.org.

### Grants and Sponsorships

**New (2018)**

- Baylor College of Medicine, Texas Children’s (one-year study); Stephen C. Mack, Ph.D. 
  *Role of MOS Proto-oncogene in H3.3K27M diffuse intrinsic pontine glioma*
- Dana-Farber Cancer Institute, Boston Massachusetts (two-year study); Brendan D. Price, Ph.D. 
  *Targeting H3.3 mutations in pediatric high grade gliomas*
- University of Virginia Cancer Center Charlottesville, (one-year study); Daniel W. Lee, M.D. 
  *Rapid Development of Chimeric Antigen Receptors (CARs) for DIPG*

**Sponsorships**

- Children’s National Foundation, Javad Nazarian, Ph.D., *DIPG Innovation Fund for DIPG Roundtable evaluations and discovery*
- *International Symposium on Pediatric Neuro-Oncology (Silver Sponsor) and Nurses Symposium June 29 - July 3, 2018*

**Second-year**

- University of Maryland, Baltimore MD, Yin Wang, Ph.D. 
  *A Novel Mechanism and Immediately-Translatable Targeted-Therapy for Group 3 Medulloblastoma*
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Surgical Theater set-up in the Oval Office to demonstrate a virtual tour of the brain. Alex, a rep for Surgical Theater demonstrated on his computer what the individual sees in 2D. Alex offered attendees an opportunity to wear the virtual reality apparatus to experience Surgical Theater’s cutting edge technology. Many attendees took a virtual reality tour of the brain in 3D.

Roger Packer provided an overview of the Foundation’s accomplishments, funding over $3.5 million for research programs that have enabled young investigators to jumpstart their research programs. He stated that many of the programs funded by CBTF have moved into significant research programs that have been beneficial to patients. CBTF funding has also supported international conferences for medical professionals and Family Retreat Day events that provide education and support for families.

Packer spoke glowingly of the honorees and provided award recognitions. All award groups received citations signed by Governor Larry Hogan and individuals received citations from Senator Christopher Van Hollen.

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Thank you to the Chairs!

Special thanks to Kyle Barker our DJ and live auctioneer. Kyle did a great job encouraging bids from attendees for the special items:
• Manoir Vacation in Tréguiuer, France
• Mervis Pearl and Diamond Necklace
• Framed photo autographed, (7 key players) Washington Capitals Team with Stanley Cup,
• Authentic Redskins Helmet autographed by over 25 players from multiple years

(continued on page 5)
Brendan Price, Ph.D., Department of Radiation Oncology, Dana-Farber Cancer Institute, Harvard Medical School

**Targeting H3.3 mutations in pediatric high grade gliomas**

Pediatric high-grade gliomas (pHGG) have few treatment options and most children diagnosed only survive for 1-2 years. Identifying new treatments is therefore of the highest priority. Work in the lab is focused on working out how these brain tumors arise. Pediatric gliomas frequently have mutations in one of the proteins which package the DNA in the cell. This protein, called H3.3, plays a very important role in controlling how DNA functions in the cell. Our work indicates that H3.3 is important for the cells ability to detect and repair damage to its DNA, and that mutation of a single amino-acid (glycine 34) in H3.3 (called H3.3G34R) blocks one of the cell’s DNA repair pathways. This loss of DNA repair has a cascade effect, leading to further genetic changes which ultimately lead to tumor development. However, the loss of this DNA repair pathway in the H3.3G34R pediatric gliomas means that the tumor cells must rely on other DNA repair pathways to grow. We propose that loss of DNA repair in pediatric gliomas expressing the H3.3G34R mutation represents an “Achilles heel” which we can exploit to develop new targeted therapies for this disease. With our new funding from the Child Brain Tumor Foundation, we plan to generate a series of pediatric glioma cell lines expressing the H3.3G34R mutation, and then use these to characterize both the role of H3.3 in DNA repair, and to carry out a small screen for potential new drugs which can specifically kill these tumor cells.

Allison Martin, MD, Johns Hopkins University Hospital

**Final Summary**

**Targeting MYC Driven Medulloblastoma with Combinatorial Immune Checkpoint Blockade**

Medulloblastoma is the most common malignant brain tumor in children. In cases of relapsed or refractory disease there is no known curative therapy. To address this question we have generated fetal mouse brain cells that mimic the most aggressive phenotype of medulloblastoma called “anaplastic” when they are placed into the cerebellum of mature mice. These cells grow tumors that look and behave like aggressive forms of medulloblastoma in humans and contain the gene mutations most commonly found in relapsed disease.

We are using this model of medulloblastoma to test combinations of immune checkpoint inhibitors. Immune checkpoint inhibitors are drugs that unleash the body's immune system defenses allowing it to seek out and eliminate tumors. Although this therapy does not always work in patients with cancer, when it does the responses are usually very good. Thus far we have found that the administration of 2 different immune checkpoint inhibitors changes the immune cells that infiltrate the brain tumors in a way that makes them more likely to be able to fight the tumors. Although, we have not seen a dramatic benefit in mouse survival overall, a small number of mice benefit from this treatment. This is not dissimilar from clinical trials of these treatments where often only a small percentage of people respond to therapy.

Importantly, we will use this information to test tumors from mice that have and have not responded to this treatment to understand how they are different. We think this will give us information that can be used to help select patients that will benefit most from this type of therapy for medulloblastoma and possibly other pediatric brain tumors in the future.

As we continue to understand these effects we want to propose a clinical trial for patients with relapsed and refractory medulloblastoma.

We thank the Childhood Brain Tumor Foundation for your support of this research, it would not be possible without you!

Dr. Yin Wang, University of Maryland, Baltimore MD

**A Novel Mechanism and Immediately-Translatable Targeted Therapy for Group 3 Medulloblastoma**

Dr. Wang's group has made significant progress in the project, “A Novel Mechanism and Immediately-Translatable Targeted-Therapy for Group 3 Medulloblastoma”. They nearly completed their studies in demonstrating that HIF1α directly binds to the MYC promoter to regulate its expression in G3MB. They have found 3 putative HIF1α binding sites on both the human and mouse MYC promoters. By gel electrophoresis, they identified a putative binding site in the mouse Myc promoter region able to bind to stabilized HIFα-pA protein. Their results demonstrated that HIF1α directly induced (continued on page 5)
MYC expression. Dr. Wang’s group is now focusing on completing their studies to identify the HIF1α-MYC axis by Crispr/Cas9 guided RNA mutagenesis of the MYC promoter in G3MB cells. Dr. Wang’s group is currently conducting studies to demonstrate the preclinical efficacy of liposomal echinomycin in group 3 medulloblastoma (G3MB) mouse models. They have analyzed the dose response of PDX1572 cells to liposomal echinomycin in vitro and found that the cells are very sensitive to the drug. They have determined that reformulating echinomycin into liposomes significantly reduced the drug toxicity compared to the previously used clinical formulation by conducting toxicity studies in mice. Dr. Wang’s group has established the mouse model of G3MB by stereotactically transplanting PDX1572, PDX-MB002, and PDX-F211 cells expressing luciferase into the mouse cerebellum, which has enabled the tumor growth to be monitored by bioluminescence imaging. They tested the therapeutic effect of liposomal echinomycin in G3MB mice using the PDX-MB002 cell line, and found that the drug increased the survival rate. They are now preparing to test the therapeutic effect of liposomal echinomycin in G3MB mice using the PDX1572 and PDX-F211 cell lines.

Baylor College of Medicine, Texas Children’s (one-year study); Stephen Mack, Ph.D.
Role of MOS Proto-oncogene in H3.3K27M diffuse intrinsic pontine glioma

My CBTF-DIPG proposal aims to evaluate the function of MOS, a candidate DIPG oncogene identified specifically in DIPG-H3K27M tumors. We recently mapped the active chromatin landscape of DIPG to uncover transcriptional dependency genes as potential targets for therapy using H3K27ac ChIP-seq profiling. V-mos moloney murine sarcoma viral oncogene homolog (MOS) was identified as highly active and transcribed specifically in H3K27M driven DIPG tumors through presence of a specific super enhancer targeting MOS. Super enhancers targeted several other MAPK pathway members including MAPK4 and MAPK10 exclusively in H3K27M tumors, along with known DIPG-H3K27M related genes such as LIN28B, MYC, and PDGFRα. Our data lays the foundation for our hypothesis that MOS and its downstream effectors MEK1/2 and ERK1/2 are required for DIPG-H3K27M tumor maintenance and represent lead targets/pathways to evaluate as a therapeutic strategy. We seek to prove our hypothesis by: 1) Evaluating the role of MOS on cellular proliferation and tumor maintenance in H3K27M mutated DIPG tumor models, 2) Leveraging transcription factor maps to understand the cis-regulatory programs that regulate MOS expression, and 3) Evaluating the efficacy of small molecule inhibitors against downstream MAPK effectors in MOS expressing DIPG-H3K27M tumors. Findings from my proposal, specifically pre-clinical in vivo testing of prioritized MAPK inhibitors lays the foundation for extension of these results to DIPG clinical trials. This goal is in line with one of the missions of CBTF to improve treatments of childhood brain tumors.

Daniel W. Lee, MD, University of Virginia Cancer Center Charlottesville, VA (one-year study), Rapid Development of Chimeric Antigen Receptors (CARs) for DIPG. A summary will be included in the next newsletter edition.

Children’s National, Foundation, Javad Nazarian, Ph.D., Msc DIPG Innovation Fund for DIPG Roundtable evaluations and discovery

The Childhood Brain Tumor Foundation was a bronze sponsor of this important roundtable meeting. The Round Table Discussion was held on October 1-2, 2018) at Children’s National. The two-day event brought together renowned scientists, clinicians, and neurosurgeons to share data, ideas, and resources to expedite effective treatments for children diagnosed with these highly aggressive and difficult to treat brain tumors.
Gala and CBTF Superheroes 2019 5K

Steve Young and the Sounds played for the evening after the live auction, and were accompanied by Kyle.

Thank you to Bridgette Wood, Shannon Falter Smith, and Mike O’Brien for volunteering their time to create the registration page and for website support.

We hope to see you next year on Saturday, November 9, 2019 at another favorite venue, the Glenview Mansion. More information will be available in a future newsletter.
On Saturday, July 14, 2018, CBTF hosted Family Retreat Day, Education and Support in partnership with Children’s National Medical Center at the Glenview Mansion in Rockville, Maryland. Special thanks to Lauren Hancock, Debbie Lafond, and Katie McHugh for collaborating to create an informative day. Family Retreat Day was admission-free and included lunch for all attendees. Childcare was provided by volunteers, allowing parents to attend sessions led by excellent experts. Children did arts and craft, took an onsite field trip to the Nature Center on the grounds, were entertained by the Blue Sky Puppet Theater, and treated to music by the boogie-woogie band Rocknoceros.

Keynote Speaker: Eugene Hwang, MD is a pediatric neuro-oncologist at Children’s National and is Associate Professor of Pediatrics and Director of the Neuro-Oncology Immunotherapeutics Program. His topic was entitled Immunotherapy and Childhood Brain Tumors.

Dr. Hwang provided a detailed review of what comprises the immune system and health matters that may develop in an immune system gone wrong. The role of immunotherapy as cancer treatment was covered, inclusive of CNS success stories. Other cancers, such as metastatic melanoma, non-small cell lung cancer, and renal cancer and their target checkpoint inhibitors were referenced. The immune system is capable of killing tumor cells. Many clinical trials are being conducted. Hwang presented information about Clinicaltrials.gov and the landscape of immunotherapy trials in pediatric brain tumors (pBTs). A graph indicated the progression of immunotherapy trials since 2000 showing the increase in trials over the past two decades which are expected to increase significantly over time.

Immunotherapy techniques include antibodies, adoptive cellular therapy, vaccines, and checkpoint blockade.

Antibodies: bind surface antigen on the tumor surface. They recognize/bind to the tumor cell and bind long enough to initiate an effector mechanism.

Adaptive cellular transfer: (ACT) involves T-cell harvest and has proved highly successful in leukemia and solid tumors. ACT is often combined with lymphodepletion (non-myeloablative). There are limitations for ACT. For instance, without the appropriate antigen the strategy will not be successful. ATC would have difficulty penetrating and difficulty retaining activity, (hypoxia, suppressor of T-cells, and immunocytokine accumulation). Dr. Hwang explained the ACT – CAR – T in humans and regression of GBM after Chimeric Antigen Receptor T-Cell Therapy. The slides showed complete response in all lesions that were treated with CART – IL13Rα2.

Vaccines may be co-administered with immunostimulatory agents and are expanding via clinical trials with a hope of better outcomes for patients. A vaccine would be comprised of cells collected via tumor biopsy, used for the individual.

Immune Checkpoint Blockade: A listing of programmed death 1 (PD1) and ligand 1 PDL1 checkpoint inhibitors that have been approved for various cancers was included. Checkpoint inhibitors have shown response in pBTs and have FDAs approval for many cancers.

Pseudo progression versus Response
Recognition of pseudo-progression (increased contrast enhancement on scan) is a challenge regarding radiologic response and how to assess eligibility for a patient to remain on a trial. Radiographic response criteria and eligibility are being revised. It can be perplexing, pseudotumor progression or treatment effect. Incidence of tumor progression can be quite variable.

Necrosis: Tumor cell necrosis, release of antigens, decrease of MDSC’s/regulatory T Cells (Tregs) can be induced by radiation. It can be difficult to determine necrosis from recurrence. Radiation necrosis can develop months out from radiosurgery or radiation usually from small vessel injury.

In conclusion, immunotherapy is changing and enhancing cancer treatment options. However, some challenges still remain. Good predictive markers are needed, fine-tuning clinical trial designs, and tumor/non tumor biological studies are needed. The CNS the blood brain barrier can be an added challenging factor.

Genetics
The second topic Genetics 101 & Brain Tumors and Genes and Cancer included co-speakers, Joyce Turner, MS, CGC and Miriam Bornhorst, MD from Children’s National. The first of the two sessions included an overview about genetics to provide a basic understanding of genetic terms; how to differentiate between genetic and hereditary; sporadic versus cancer predisposition; understanding genetic testing, and understanding what a genetic evaluation entails.

(continued on page 9)
Ms. Turner explained that mutations change the “spelling” of genes in a harmful way. The four types of chemical gene structures are known as nucleotides are labeled Adenine (A), thymine (T), cytosine (C), and (guanine) G. Molecular testing is important for diagnosis, prognosis, treatment, and for guiding germline testing (at times). Predisposition to cancer syndromes can be revealed, the cause of cancer, screening, interventions, and beneficial treatment agents.

**Pediatric Endocrinology – Endocrinopathies and Fertility in Cancer Survivors**
Christiana Tasti, MD, Ph.D. is a fellow in pediatric oncology at the National Cancer Institute at the National Institutes of Health. Forty to 50% of cancer survivors will develop at least one endocrinopathy over their lifetime. Pertinent factors include, age, gender, genetic background, disease and location of tumor, and treatment regimens. Radiation exposure is key factor for endocrinopathies. Cancer treatment and location are significant. Thyroid function, cortisol production, growth failure, growth hormone deficiency and management are common endocrinopathies. Risks and concerns include increased intracranial pressure, edema, slipped capital femoral epiphysis, worsening of existing scoliosis, gynecomastia, hyperglycemia, and recurrent cancer or second malignancy. Tasti discussed reproduction and fertility concerns for cancer patients, such as, precocious puberty, hypogonadism, (lack of sex hormones). Fertility preservation guidelines are available and a consideration for cancer patients and can be underutilized resources for assorted reasons.

**Neuropsychology and School**
Kristina Hardy, Ph.D., neuropsychologist, faculty position in the Neuropsychology program at Children’s National, provided a detailed overview about executive functioning and the deficits it may cause in pediatric brain tumor patients. Executive functioning is important in helping regulate behavior, thinking and emotions. Other diagnoses often carry the same problems, such as ADHD, autism, and anxiety. Research related to other diagnoses can provide additional treatment strategies. School-based problems and anxieties difficulties may develop. Hardy explained some of the impact related to functioning in different domains, home school, social, and interpersonal. Keeping track of possessions, following through on directions, problems with multi-step directions, attention span, and lack of awareness of their difficulties are some of the problems.

In summary, at times, children with executive functioning problems exhibit performance deficits rather than skill deficits. Treatment is needed, without, performance problems can lead to problems with acquisitions of skills. Dr. Hardy also covered school-based interventions.

**Integrative Therapies**
Deborah Lafond, DNP, PPCNP-BC, CPON, CHPPN, Children’s National; Elizabeth Bettini, Ph.D., PCNS-BC, CHPPN; Lauren Cates, CMT, and Risi Idiokitas, L.Ac., DAOM
Integrative therapies are appropriate for children, adolescents, and young adults with serious illness to promote comfort and optimize their quality of life. Non-pharmacologic pain and symptom management options may include: parental presence, visualization or guided imagery, deep breathing, massage, positioning, music, Reiki, play therapy, aromatherapy, acupressure, and mindfulness training. These approaches can be beneficial for stress reduction. Neonates and infants would require different techniques, such as, swaddling, infant massage, minimization of sleep interruption, oral sucrose, and music. Consideration for integrative therapies was reviewed and discussed with attendees.

Pamela L. Wolters, Ph.D., psychologist at the Pediatric Oncology Branch of the National Cancer Institute, National Institutes of Health is conducting a research study for children with cognitive late effects, the PACE Study. The study involves a short evaluation, fitness testing, and a blood draw. If you would like to learn more contact Dr. Pam Wolters, (wolters@mail.nih.gov or call 240-760-6040.

Lauren Hancock, RN, MSN, CPNP-AC, Neuro-Oncology Nurse Practitioner, Children’s National closed out the day leading a discussion covering family perspectives and engaging our attendees.

Thank you to all of the nurse practitioners that helped plan such great program. We appreciate the time each speaker gave voluntarily to make the day a wonderful opportunity for families.
GRACIOUS GIVING, THANK YOU FOR YOUR SUPPORT!

Thank you to our friends who donated through workplace charitable giving campaigns this year, inclusive of the CFC, Maryland Charity Campaign and other independent campaigns.

The Childhood Brain Tumor Foundation, friends and families are very grateful of your support.

Maryland Charity Campaign

Campaign donations may be made for the United Way (UW) through the “donor option” or “donor choice.” Please check with your employer in reference to UW campaigns. You may write us in as your choice.

The Childhood Brain Tumor Foundation participates in the Combined Federal Campaign (CFC) and Maryland Charities. Check our Website in the late summer or fall for our confirmed designated numbers.

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Our website: www.childhoodbraintumor.org

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The Childhood Brain Tumor Foundation is dedicated to funding research for all pediatric brain tumor types. Please contact us if you have any questions:

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We need your support.

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We welcome any donations in the way of financial contributions and sponsorships.
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