# Meeting the Editorial Board Member of *PRPM*: Prof. Kenneth Blum

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## Expert introduction

Dr. Kenneth Blum received his Ph.D. in Neuropharmacology from New York Medical College and graduated from Columbia University and New Jersey College of Medicine. He trained at the Institute of Behavioral Genetics, Colorado University at Boulder, Colorado. Dr. Blum also received a Doctor of Humane Letters from St. Martins University. He has published over 675 abstracts, peer-reviewed articles, and 17 books. Currently, with Dr. Thomas McLaughlin, who is Harvard-trained, he is completing a book entitled "Death by Neuron."

He has been the recipient of many grants and awards, including a Life-Time Achievement in Addiction Medicine (Holistic Institute on Addiction Studies); Marquis Who Who's Life–Time Achievement Award, and Presidential Award for Scientific Excellence (National Council of Alcohol & Drug Abuse Councilors), Scientific Achievement Award (City of Life Miami) and Best Abstract (2012) Award ASRA (Pain); Path Foundation Lifetime Achievement Award (2014) and Honorary Full Professor (Eötvös Loránd University, Institute of Psychology, Budapest, Hungary) and American Society of Addiction Medicine Millennium Laboratory Award and Top Registry Professional of the Year Award 2018 among many others. He coined the term "Reward Deficiency Syndrome" in 1995, now in Microsoft Dictionary, Gates Scientific dictionary, and featured in SAGE Encyclopedia of Abnormal Psychology (2017). Selected Marquis Who's Who Scientist of the Year (medical) 2019 and Marquis Who's Who in the world (2021). Featured researcher in Scientia (2022).

Currently, he serves as Editor-in-chief (EIC) of *Journal of Reward Deficiency Syndrome* & *Addiction Science* (Editor-in-chief), EIC of *Journal of Systems* & *Integrative Neuroscience*, EIC of *Current Psychopharmacology*, EIC of *Journal of Addiction* & *Recovery*, and Co-EIC of *Journal of Neuroimaging in Psychiatry* & *Neurology*, Regional Editor of *Current Pharmaceutical* & *Biotechnology*, and Section Editor (Neurogenetics and Nutrigenomics) and Current Psychiatry & Research and Reviews and is on the editorial board of 18 other scientific journals. As the lead author on the first genetic association of a dopaminergic gene with severe alcoholism, he is considered by some as the "Father of Psychiatric Genetics." He is the holder of many US and foreign patents involving nutrigenomics. Recently he has been awarded the USA patent on Genetic Addiction Risk Score (GARS). Dr. Blum received 22 patents on GARS across Europe. He is the lead author of the Springer Neuroscience Brief book on the 12 Steps—entitled "Molecular Neurobiology of Addiction Recovery." Dr. Blum and Mark Gold (St. Louis) have been named Editor-In-Chief of *Frontiers of Bioscience* Special Issue on Reward Deficiency Syndrome (RDS). Dr. Blum was a Volunteer Professor in the Department of Psychiatry at the University of Florida College of Medicine and McKnight Brain Institute. Currently serves as adjunct Full-Professor in the Department of Psychiatry at the University of Vermont. He was an Adjunct Full Professor in the Department of Psychiatry & Behavioral Science, Keck School of Medicine, University of Southern California. He is currently a Clinical Full Professor in the Department of Psychiatry, Wright University Boonshoft School of Medicine, Dayton, Ohio. He is currently serving on the research faculty of Western University Health Sciences, Pomona, California. He also served as Neuroscience advisor to many companies and foundations, including Dominion Diagnostics (Chief Scientific Adviser), Path Foundation NY (Director of Science), Victory Nutrition, Impact Genomics, Shores Treatment & Recovery Center (Neuroscience Advisor). He also served as Chief Scientific Officer and is the Emeritus Chairman of Geneus Health and Restoregen, San Antonio, Texas. He is Emeritus Faculty of The Institute of Applied

Genomics and Biotechnology, Nagpur, India. Major media outlets worldwide have covered his work on addiction. Dr. Blum was Chief Neurogenetic & Addiction Therapy Advisor, The Florida House Experience. He is currently serving as Founding President of United Science Group (USG).

Dr. Blum has developed the first-ever patented GARS with Geneus Health. He was a frequent contributor to Sober World and Addiction Professional magazines (no longer published). In conjunction with Merlene Miller and David Miller, they just published their award-winning book "Staying Clean and Sober." He is a sought-after speaker on a global basis for insights into RDS and genetics. He has published in almost every major peer-review journal globally, including *Science*, *Nature*, *Lancet*, *JAMA*, *JAMA Psychiatry*, *PNAS*, *Plus One*, *Oncotarget*, *Cureus*, *Psychopharmacology*, *Current Neuropharmacology*, amongst many other prestigious journals. Every major newspaper, magazine, television network worldwide, and the Harvard Review featured his work. He is indeed the father of Amino-Acid Therapy for Reward Deficiency Syndrome developing Pro—Dopamine Regulation to induce "dopamine Homeostasis "coupled with genetic testing for "Precision Addiction Management" (PAM®) and Precision Behavioral Management" (PBM®). He also has vast business experience, including former Chairman of a Public Company. Most recently, with Dr. Marjorie Gondre-Lewis Howard University and the National Human Genome Center (NHGC), he was Principal Investigator of a grant from the NIH selected to be funded related to both GARS and Pro-dopamine regulation in the African-American population along. He is a nominee to the National Whole Genome Science Foundation board of directors. Currently, he serves as Executive Chairman of InGeneUS Inc. & Chairman and CSO of The Kenneth Blum Behavioral & Neurogenetic Institute (Division of iVitalize, Inc.). Dr. Blum along with Dr. Eric R Braverman serve as EIC for the special issue on Global Addiction for the *International Journal of Environmental Research & Public Health* with associate editors Dr. Mark Gold, David Baron and Jean Lud cadet.

## Interview

***PRPM: What do you think is the role of pharmacogenetics in psychiatry treatment?***

**Prof. Blum:** The role of genetics in psychiatry is emerging, with thousands of studies pointing to the identification of DNA genetic polymorphisms and epigenetic insults. For example, a current word search in PubMed using "Psychiatric Genetics" resulted in 26,369 articles from 9-22-21. Many of these articles are reviews and not original studies; however, this body of information is indeed significant. It is also true that many candidate genes or even genetic clusters (GWAS) found to associate with any number of RDS addictive behaviors have relatively low Odds Ratios with small effect-size contributing to the disease in question. It is indeed a complex array consisting of a polygenic score. My team pursued relevant information for genetic testing for precise pro-dopamine regulation to induce "dopamine homeostasis", especially in people carrying an array of DNA risk polymorphisms from across the brain reward circuitry. In addition, there is evidence to support the concept of Reward Deficiency Syndrome (RDS), the rationale for risk assessment for vulnerability to both substance and non-substance addictive behaviors. PGX is important primarily to provide precision medication dosing because there are many individualized polymorphic differences, especially as a function of ancestry. However, the future requirement is to provide a genetically guided basis for therapeutic delivery across all psychiatric disorders like rapid treatment of depression to attenuate suicidal ideation.

***PRPM: Would you like to give us a general picture of the publication area in pharmacogenomics? Any topics or papers that impressed you most in the past two years?***

**Prof. Blum:** In terms of pharmacogenomics, I believe there is a lack of good genetic research. Specifically, the issue here is that many studies do not utilize appropriate controls. We can't expect accurate results if we analyze a control that has the disease present. It is akin to evaluating disease *vs.* disease, and as such, the results are likely spurious. This lack of appropriate controls is also present in studies related to reward processing and associated SUD or PGX, including non-SUD behavioral addictions, which contain many hidden addictive behaviors. My laboratory has proposed 'Supper Controls' highly screen cohorts that exclude all Reward Deficiency Syndrome (RDS) addictive behaviors and disorders. Unfortunately, even in GWAS's highly sophisticated large population studies, this suggested solution is not yet adopted. Also, there are many associated loci and gene alleles obtained with GWAS studies that may reflect, in many cases, second messenger proteins, which certainly have relevance for the predictability of a specific type of addictive behavior. Still, it seems prudent that convergence to candidate genes as finite reward circuitry

 

endophenotypes is necessary to help develop a Genetic Health Risk test a polygenic score for professional field and consumer benefit. There are too many papers in the field to pick just a few. However, two recent papers that have supported the original work on the *DRD2 Taq A1* allele we reported in *JAMA*, 1990 (1-3).

***PRPM: What originally leads you to the study of addiction medicine and genetics?***

**Prof. Blum:** That is a very loaded question. Because there is no simple answer and over the sixty years of researching the area, addiction medicine and especially genetics have changed remarkably.

In the 1970s many scientists, especially psychiatrists like Drs Donald Goodwin and Mark Schuckit worked in Sweden with a large cohort of twin data. They suggested that if twins are separated, one to an alcoholic family, and the other to a non-alcoholic family, it did not matter if they were raised by a non-alcoholic parent or an alcoholic parent; if they carry the genetic propensity, they could become alcoholic. This work suggested that separated twins were vulnerable to becoming alcoholics despite the role of upbringing.

In the late 60s, I was awarded a grant from the NIAAA institute to study the role of stress and alcoholism. From that moment on, I have been "addicted" to this area of research. In 1978 I completed a fellowship in animal genetics at *Colorado University Institute of Behavioral Genetics* with Dr. Gerold McClain, the father of animal genetics. Following the completion of that fellowship, I have dedicated my laboratory to the genetics of alcoholism. The most prominent study was published in Proceedings of the National Academy of Sciences (PNAS); we showed that alcohol intake is related to the quantity of endorphins (met-enkephalin) in various regions of the rodent brain. We found that the lower the amount of met-enkephalin, the higher the alcohol intake based on genetically bred mouse strains (4). One additional exciting finding was the fact that when I increased the quantity of met-enkephalins in these genetically bred alcohol loving mice (C57/BL) using a substance called D-Phenylalanine, which prevents the breakdown of the met-enkephalins by a carboxy-peptidase enzyme, a pharmacogenetic engineering result was induced and the C57 black mice now drank lower amounts of alcohol like non-alcohol preferring DBA mice (5).

During the 80s, the field was buzzing with the question, "how are we ever going to find the first gene to associate with alcoholism?". Understanding this most perplexing question in the field, in 1987, I decided to crack this mystery. I called my dear friend Dr. Ernist P Noble (deceased former director of the NIAAA) outlined a plan to explore the reward circuitry genome to pinpoint a specific gene to associate with alcoholism.

In 1990, our laboratories UCLA and University of Texas published our seminal finding in *JAMA* associating the *DRD2 TAQ* A1 allele with severe alcoholics relative to controls. Besides the global media impact, scientifically, it opened the gate to the robust area of "*Psychiatric Genetics*" (1). While controversial at the time, that particular gene has been the subject of over 6,000 studies. *The rest is history.*

***PRPM*: *During research., what are the biggest challenges/ problems you had faced? And how would you sort them out?***

**Prof. Blum:** Not only is the question related to the biggest problems I have had to face in terms of my research, in my sixty-year sojourn, limited to describing a few examples, is somewhat tricky because there have been many. During this journey, I have published 675 articles, book chapters, and professional meeting abstracts, along with 18 books. However, with that stated, a few research studies stand out from a large body of work.

*(I) Alcohol inhibits the synthesis of endorphin* (6)

As a participant in the first Gorden Research Conference on Opioid Peptides and applauding Dr. Eric Simon of NYU for naming these unknown brain opioid peptides (a contraction of endogenous morphine), calling them endorphins was a most exciting time for me. On the heels of this excitement, working in alcoholism, my lab was the first to report that long-term alcohol consumption (20 years of the human equivalent of alcohol usage) in golden Syrian hamsters that love to imbue alcohol revealed the interrelationship of alcohol and brain endorphins. The challenging aspect of this investigation involved the exact measurement of the amount of alcohol compared to water consumed by these hamsters. To obtain an accurate amount of alcohol, my staff and I measured this consumption at precisely 4 am for 360 days (with no days off). In fact, from Friday through Sunday, I had to leave home and go to the laboratory by 4 am to measure the amount of alcohol intake consumed by the hamsters relative to the amount of water consumed

 

by the controls. The most challenging part of this experiment was to explain to my wife that I was not having an affair. However, I was vindicated by publication in *Science* magazine in 1982.

This experiment's take-home message is that long-term alcohol drinking inhibits endorphins, particularly enkephalin. Only perseverance helped me sort this out.

*(II) Finding the first gene to associate with severe alcoholism* (1)

In 1987 following a visitation from Dr. Noble and receiving a half a million-dollar grant from the Seavers Foundation in California, we embarked on a challenging nightmare. At that time, genomic scientists believed that there were one-hundred-thousand functional genes in the human genome. Like finding a needle in a haystack, we needed to find one out of a hundred thousand.

Before 1990 the genetic tools to find associated genes with diseases were very slim. There was no understanding of computational analysis and GWAS to identify polymorphic gene alleles. Utilizing one bacterial endonuclease per allele encompassing 29 possible bacterial endonucleases for one hundred thousand genes was quite a task. After exploring seven likely genes and associated alleles using only one bacterial endonuclease, we failed in finding any evidence for an associated genetic polymorphism. Once again, expressing my deep frustration to my family, I suggested that I would be a thousand years old before finding this "needle in a haystack." However, with encouragement from my family and scientific colleges, we resolved to continue the pursuit. Two years later, Dr. Noble and I received a congratulatory call from the excited editor-in-chief of *JAMA*, letting us know they would publish our article on April 18th, 1990. In our paper, we ensured that the DRD2 TAQ A1 allele was not specific to alcohol perse but to all reward deficiency behaviors. The main reason our joint research was able to sort out this conundrum was primarily that along with Dr. Gerald Kozlowski from South-Western Medical School in Dallas, Texas; we had developed the brain reward cascade, which acted as our blueprint to identify neurotransmitter genetic associations (see *Figure 1*) (5).

*(III) Coining the term Reward Deficiency Syndrome (RDS)* (7)

This concept was indeed very challenging. In actuality, my concern over the many years in the addiction medicine space was the enormity of the scientific information emerging from all parts of the world. In 1995 I realized that there was no unifying nosology that captured the diversification and array of all the substances (alcohol, opioids, stimulants, sedatives, nicotine, psychedelics, and glucose) and non-substance addictive behaviors and psychopathology (gambling, over-eating, and other eating disorders like anorexia/bulimia, ADHD, PTSD, shopaholics, workaholics, etc.). The issue related to scientific meetings that were classified according to individualized substances or addictive behaviors. However, the biggest challenge was to come up with a unifying name to describe this far-reaching concept. The challenge was to either ascribe a surfeit or deficit of various reward circuitry neurotransmitters, prominently dopaminergic. As discussed above, the brain reward cascade (as in *Figure 1*) depicts the interrelatedness of several neurotransmitter systems, including serotonergic, cannabinergic, gluconeogenic, endorphinergic, GABAergic, glutaminergic, cholinergic, culminating with the net release of dopamine at the nucleus accumbens. Based on the literature at that time, substance-seeking behavior increased with a deficit of dopaminergic function, and I observed hypodopaminergia as opposed to hyperdopaminergia. Then I copied the idea of Auto-Immune Deficiency Syndrome (AIDS), and that led me to the name Reward Deficiency Syndrome (RDS). In reality, there is now evidence that, for example, teenagers may have a neuro-epigenetic developmental hyperdopaminergia and as such obtain a stronger "high" when they imbibe in alcohol and other drugs as compared to adults. Importantly there is emerging genetic evidence to support the RDS concept from GWAS studies globally (in multi-millions of patents). These studies show substantial genetic overlap between substance and non-substance addictive and psychopathological behaviors. This phenomenon is not the case for several neurological-based disorders such as Alzheimer's, Multiple Sclerosis, Parkinsonism, Migraine, whereby the genetic architecture is

 

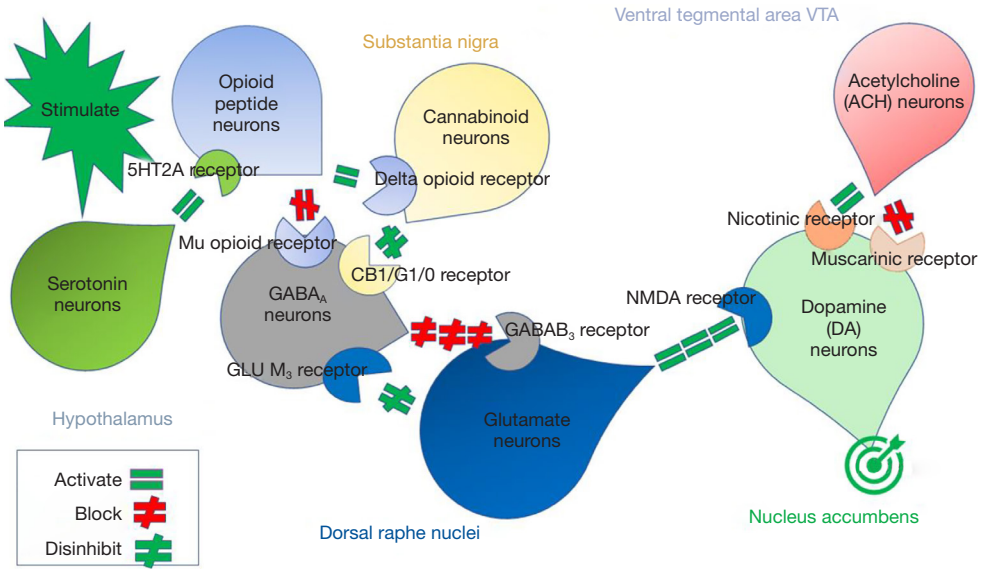


**Figure 1** illustrates the interaction of at least seven major neurotransmitter-pathways involved in the Brain Reward Cascade (BRC). In the hypothalamus, environmental stimulation results in the release of serotonin, which in turn via, for example, 5HT-2a receptors activate (equal green sign) the subsequent release of opioid peptides from opioid peptide neurons, also in the hypothalamus. Then, the opioid peptides have two distinct effects, possibly via two different opioid receptors. One that inhibits (red hash sign) through the mu-opioid receptor (possibly via enkephalin) and projects to the Substania Nigra to GABAA neurons. Another stimulates (equal green sign) cannabinoid neurons (the Anandamide and 2-archydonoglcerol, for example) through Beta –Endorphin linked delta receptors, which in turn inhibit GABAA neurons at the Substania Nigra. Also, when activated, cannabinoids, primarily 2-archydonoglcerol, can indirectly disinhibit (red hash sign) GABAA neurons through activation of G1/0 coupled to CB1 receptors in the Substania Nigra. In the Dorsal Raphe Nuclei, glutamate neurons can indirectly disinhibit GABAA neurons in the Substania Nigra by activating GLU M3 receptors (red hash sign). GABAA neurons, when stimulated, will, in turn, powerfully (red hash signs) inhibit VTA glutaminergic drive via GABAB 3 neurons. It is also possible that stimulation of ACH neurons at the Nucleus Accumbens ACH can stimulate both muscarinic (red hash) or Nicotinic (green hash). Finally, Glutamate neurons in the VTA will project to dopamine neurons through NMDA receptors (equal green sign) to preferentially release dopamine at the Nucleus Accumbens, shown as a bullseye that indicates a euphoria or "wanting" response. The result is that when dopamine release is low (endorphin deficiency), unhappiness is felt, while general (healthy) happiness depends on the dopamine homeostatic tonic set point (with permission from Blum).

distinctive. Simply there are shared inheritable genetic antecedents across all psychiatric disorders. This concept flies in the face of the DSM-V, which provides symptomology, not etiology.

*(IV) Genetic addiction risk severity test (GARS)* (8)

Following the seminal research in which Noble and Blum in 1990 the first genetic association of the DRD2 TAQ A1 allele and severe alcoholism, despite its controversy in the field, we realized that this polymorphism was not specific for an alcoholism endophenotype but was a non-specific reward deficit allele. After 14 years and a large number of candidate gene association studies for all RDS behaviors, and many genetic polymorphic alleles were found, I developed a novel panel of ten genes and eleven alleles that captures the primary neurotransmitter induction of a hypodopaminergia. In a study involving over four hundred people that attended chemical dependency programs found that individuals carrying four or greater of these alleles or carrying seven or greater of these alleles had vulnerability or risk for either drug or alcohol dependence, respectively. This work resulted in the USA-issued patent in 2020 and twenty-two other foreign patents. To sort out the important utility or clinical applications of the GARS test, we published studies related to alcohol and drug vulnerability, utility in identifying high-risk opioid pain patients

and GARS test utility in bariatric surgery.

(V) *Precision behavioral management* (9)

To sort out the challenge of appropriately utilizing the GARS test, we decided to develop Precision Behavioral Management (PBM). This technique enables the coupling of the genetic antecedents to seeking-behaviors and addiction vulnerability, based on either high or low dopamine function. The benefit of coupling the GARS test with pro dopamine regulation (KB-220, a nutraceutical complex) utilizing precision algorithms enables "dopamine homeostasis" (10). One example that has been published from our laboratory yielded significant benefits to a family having RDS behavioral issues (binge drinking, substance abuse disorder, anger, etc.). Utilizing PBM, in this case, resulted in three different KB220 variants. With significant beneficial clinical outcomes in the initial proband (daughter, mother, and father). The daughter is consistently utilizing KB220 and has been staying clean and sober, the father is no longer binge drinking, and the mother has attenuated anger.

***PRPM: Can you tell us about any new and exciting projects you're working on? What do you foresee as the next step in your research journey?***

**Prof. Blum:** While our group continues to develop new applications for PBM, such as utilizing the GARS test in Neonatal Opioid Withdrawal Syndrome (NOWS) and other unwanted behavioral sequalae, our new and exciting project relates to the possibility of "curing" RDS behaviors. This new project involves gene editing to help ascribe normative functioning of messenger RNA (11).

***PRPM: You have built quite a career, your research, your profession, and your mentorships etc. How do you combine all this? Where did you get your energy?***

**Prof. Blum:** The answer to this question could be very generic-my drive and passion.

However, there is more to the story. Laying in my bed at the age of eleven, I asked myself, what is the purpose of my life on earth? I contemplated the idea that I would get an Oscar for acting, receive a Pulitzer Prize for writing a book, or be awarded the Nobel Prize for Medicine and Physiology (Still working on it). In terms of my energy and drive, I believe my formative years growing up in Brooklyn, New York, where I embraced sports, especially basketball, with competitiveness and passion that resulted in being named captain of the All-New York City basketball team. The experience taught me that my passion for research later on in life was nothing more than doing my best and realizing I had to overcome hurdles and competition with an abundance of energy.

***PRPM: As an Editorial Board Member of PRPM, what is your expectation for PRPM?***

**Prof. Blum:** My expectation for *PRPM* is for them to continue to publish only high-quality peer reviewed articles that contribute to the scientific field at large. However, they should not interfere with the scientific progress by choosing sides in a controversial topic, negative or positive, because that would impede progress in both precision research and precision medicine. In today's world of scientific publishing, especially concerning payment, fees linked to open access may be a real deterrent to individual scientists who are not backed by large supportive grants or institutions. Non-payment should not be a reason to prevent the publication of scientifically accepted manuscripts.

***PRPM: PRPM is adherence to high publication standards and transparency from the start (https://prpm.amegroups.com/article/view/51187/pdf). Would you like to share your perspectives at this point?***

**Prof. Blum:** My perspective on this matter is that transparency is the best medicine. However, for example, in a number of other journals, the interactive platform is both time-consuming and frustrating. While I do not have a sound answer for this conundrum, the publishing experts must at least turn their attention to providing a better way.

***PRPM: What would be your advice for early career researchers?***

**Prof. Blum:** I suppose after being a researcher in this field for over sixty years I feel honored but humbled to give advice to anyone. In my early days as a young researcher myself, I had the opportunity to ask Nobel Prize winners Von Euler and Julius Axelrod for their advice about winning the Nobel Prize. Von Euler stated "serendipity," and

 

Axelrod said, "be a visionary." While at that time these words did not translate in terms of my everyday work, I believe in retrospect that extraordinary research occurs when serendipity, vision, and perseverance come together. There are three kinds of scientists: (I) The visionary-building on previous scientific dogma and hypothesizing something that is unseen and determining its viability and making huge leaps even without much evidence; (II) The detailer-understanding and appreciating the visionary process but not accepting its validity and performing a series of scientific experiments to fill in the missing details; (III) The naysayer-a scientific skeptic that does not appreciate the visionary hypothesis and does not attempt to evaluate through scientific method its validity, but inappropriately denounces the hypothesis without any evidence whatsoever.

My advice would be in terms of experimentation is to be accurate, do not forsake it until you prove yourself that it is wrong.

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