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Genetic Networks in Cognition, Addiction, and Homeostasis

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ABSTRACT: Genetic networks regulate cognition, addiction, and homeostasis by modulating human brain activity. In this paper, we will analyze four genetic networks - the mesolimbic system, acetylcholine network, CREB-dependent gene regulatory network, and NF-κB networks. We will analyze their roles in cognition, addiction, and homeostasis in humans, and look into how these networks relate. We will then go deeper into NF-κB pathways and analyze each of its 5 subunits to derive the common areas across the human body that are influenced by the genes interacting with those subunits. These common areas would be in terms of KEGG (Kyoto Encyclopedia of Genes and Genomes) and REACTOME pathways, disease pathways, and other biological processes. These will be termed as just *biological processes*. This derivation will be laid out in the form of Python code that can be reused to derive those common areas using the latest data from the trusted data sources. I have derived the specific genes that interact with each of the 5 NF-κB subunits and have found the biological processes they enrich. Recognizing genetic networks and their associated biological processes may help identify areas for further research or discover potential therapeutics for cognitive, addictive, or homeostatic diseases.

KEYWORDS: Cellular and Molecular Biology, Genetics, Cognition, Addiction, Homeostasis.

Introduction

The human body has around 19,000 to 25,000 genes, but the total number of genetic networks is still unknown. Genetic networks are defined by genes that control a similar process and can be categorized by cell type or purpose. They influence specific properties of the body, including cognition, addiction, and homeostasis. Cognition is the brain's ability to process and react to information and includes attention, decision-making, memory, and speech. Genes code for proteins that promote cell communication and synaptic plasticity, allowing neurons to have stronger bonds and stronger neural pathways.[1-4] These neurons and neural pathways are often activated when one is learning. Thus, stronger connections between them promote stronger memory and quicker responses to new stimuli, creating stronger cognition. Addiction is considered a chronic brain disease that happens when one abuses the use of a substance and is commonly classified into three main stages: the binge/intoxication phase, the withdrawal/negative stage, and the preoccupation/anticipation phase.[5,6] It is identified as a deficit in reward and dopamine pathways.[2,7,8] Addiction leaves problems in reward-based learning and memory that are usually permanent. Homeostasis is defined as how your body can self-regulate through external changes or stimuli.[2] The process uses feedback systems to maintain the body and ensure that activities such as temperature regulation, pH balance, and the proper management of specific elements are properly regulated.

It is vital to understand how genetic networks function under specific circumstances and in certain contexts to maintain the health of the brain and the rest of the body. Sometimes, there are issues in the brain that cause diseases across the body. If we research genetic systems and their effects, we can find target areas in the biological processes that are commonly susceptible to the underlying genes. This paper overviews four important genetic networks- mesolimbic, acetylcholine, CREB-dependent, and NF-κB - and their effects on cognition, addiction, and homeostasis. It then contrasts their roles and demonstrates a way of finding relationships within the networks.

Methods

The genetic networks considered in this study are the Mesolimbic System, the Acetylcholine Network, the CREB-dependent gene Regulatory Network, and the NF-κB Pathways. Existing research around these four genetic networks has been studied to derive their influence areas. With the listed areas, one genetic network, NF-κB, is chosen to understand the dynamics of the impacted pathways, considering the genes involved. In my research, I have used publicly available relevant data and have created the necessary Python code to surface the top biological processes enhanced by the genes relevant to NF-κB pathways in humans. This is achieved via 2 broad steps. First, I derive the gene names that interact with the NF-κB subunits. We will get 5 sets of genes, as there are 5 subunits of the NF-κB family. I then find the biological processes where those genes play a statistically significant role. Each set of genes influences a set of biological processes, and we consider only those where the p-value is less than 0.05 to ensure a higher degree of statistical significance. Uniprot is being used here as the data source since it is an open-source library of data relevant to NF-κB subunits (listed as proteins) and the genes that interact with their dependencies. This library collates and maintains experimental data from several trusted sources like IntAct, MINT, STRING, etc., and allows an API-based interface, which means we can acquire the latest data from them every time we run the code. GProfiler is used to derive the top

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relevant biological processes enriched by the genes interacting with each of the NF-κB subunits above. KEGG and REACTOME are major sources of biological processes referred to in this case. The Python code for the above work has been made publicly available here: **https://github.com/1Riya-Shukla/NF-kB-Pathways**

Results

Mesolimbic System:

The mesolimbic system is an important dopaminergic genetic network. It plays a key role in the brain's dopaminergic reward pathway.[7,8] It is found in the midbrain and works to connect these brain regions to the ventral tegmental area (VTA). The VTA contains neurons that produce and send dopamine, a neurotransmitter, across the brain in reaction to external stimuli associated with pleasure.[2] This pathway drives individuals toward goals and behavior repetition, and may even strengthen connections between cues and rewards.[7,8] A study found that defeated rats showed increased phasic dopamine signaling, a short-term boost of dopamine release during aggressive interactions, suggesting a link between social stress and dopamine activity.[8] Thus, the mesolimbic system can perform cognitive tasks by using dopamine to regulate motivation, learning, and decision-making.[2,7] Overuse of harmful substances or behaviors can cause excessive dopamine depletion from the VTA, causing the desired and addictive feeling.[7,8] Over time, the brain misinterprets the greater dopamine production from substances as normal by making fewer rewards. As a result, such substances and behaviors appear less satisfying. Now, the only way an addicted person can get more dopamine is from external direct sources like the addictive drugs that are known to deteriorate their brain.[6,7] This causes withdrawal symptoms and the constant need for a substance after it is suppressed. The mesolimbic system stays regulated by homeostasis, balancing motivation, reward, stress, and other physiological needs.[2] Genes like DRD2 (dopamine receptors) and GABRA1 (GABA receptors) affect how these signals work, making sure rewards and motivation adjust to the body's needs. If homeostasis is disrupted, genetic differences can change how these neurotransmitters function, leading to problems with impulse control, stress, or addiction. For example, when someone is hungry, their mesolimbic system will trigger dopamine levels to rise, promoting food-seeking behavior and making the idea of eating more appealing. Dopamine and other neurotransmitter signaling play a crucial role in each of these functions, ensuring motivation, learning, and behavior are properly maintained.[2,7]

Acetylcholine Network:

The acetylcholine network is a complex system that plays a crucial role in modulating neuronal excitability, synaptic transmission, and network dynamics in the brain. Acetylcholine (ACh) modulates neuronal excitability through the inhibition of potassium currents and the activation of specific muscarinic and nicotinic receptors on cell membranes, leading to enhanced synaptic communication and dynamic network modulation.[9] The acetylcholine network helps in thinking, learning, memory, and focus by improving communication between brain cells. Improved communication allows ACh signaling to be faster, stronger, and less susceptible to distractions. This makes cognitive functions stronger and more efficient.[9,10] ACh is capable of stimulating dopamine release, which strengthens substance craving. In nicotine addiction, nicotine directly binds to nicotinic acetylcholine receptors, mimicking ACh. This overly stimulates the reward system, leading it to release abundant amounts of dopamine. Eventually, the brain is more susceptible to triggers, cravings, and relapse.[5] ACh excites neurons to promote memory, but controls inhibition to prevent any overstimulation. It regulates the amount of dopamine and serotonin to balance mood and behavior and synaptic plasticity for neural communication. Specific genes are used for their purposes in ACh networks. For example, CHAT (choline acetyltransferase) produces ACh, ACHE (acetylcholinesterase) codes an enzyme that breaks down ACh, transporter genes that create proteins to move ACh, and receptor subunits that improve synaptic transmission.[9,10] In all of these functions, neural plasticity is regulated. Neural plasticity, regulated by ACh, is essential for cognition, addiction, and homeostasis by ensuring neurons adapt efficiently without becoming unstable.

CREB-Dependent Gene Regulatory Network:

The CREB-Dependent Gene Regulatory Network is a system of genes where the transcription factor CREB (cAMP response element-binding protein) regulates multiple gene expressions, specifically by binding to cAMP. The main neurotransmitter for this network is glutamate.[1,3] Glutamine controls CREB activation by producing glutamate, exciting neurons for learning and memory, and GABA, which calms them to prevent overstimulation.[1,3,5] In cognition, CREB is essential for memory formation and learning. Neurotransmitters like glutamate, acetylcholine, and dopamine activate CREB, which strengthens neural connections and supports synaptic plasticity.[2] Activation of CREB can turn short-term memory into long-term memory. Substance abuse overactivates CREB, leading to changes in gene expression, promoting further addictive behavior. When the use of a drug stops, CREB levels also drop, creating withdrawal and cravings.[4,6] In homeostasis, CREB helps maintain neural stability by regulating responses through neurotransmitters like serotonin, acetylcholine, and GABA.[1,9,11] It can adjust neuronal activity to prevent overstimulation or underactivity, creating proper cognitive and emotional balance.

NF-κB pathways:

NF-κB is a group of transcription factor protein complexes. They play a major role in neurotransmitter signaling, synaptic plasticity, and neuroinflammation. NF-κB pathways regulate inflammation in cells and act as a master switch in response to stimuli, specifically stress or infections. In cognition, activated NF-κB helps neurons adapt by monitoring genes in learning, memory, and synaptic plasticity. Whereas in addiction, NF-κB is influenced by dopamine and glutamate signaling in the brain's reward system. When exposed to addictive substances, dopamine release triggers NF-κB activation, leading to chang-

es in gene expression that reinforce drug-seeking behavior and cravings. Over time, NF-κB strengthens reward-related pathways, making addiction more persistent. Additionally, chronic drug use increases neuroinflammation, which further dysregulates NF-κB activity, contributing to withdrawal symptoms, anxiety, and relapse. Drugs like opioids, nicotine, and cocaine are known to over-activate NF-κB, leading to long-term alterations in brain plasticity. NF-κB helps maintain homeostasis by regulating the brain's response to stress, inflammation, and immune signals by adjusting neuronal activity. Chronic stress may cause persistent NF-κB activation, which disrupts mood regulation and can cause neurodegeneration.[6]

Biological processes related to NF-κB pathways:

Because there are thousands of genes in humans, categorizing each one into a specific biological process is time-consuming and cumbersome. However, with new technological advances in biological and computational fields, we can break down the problem into simpler steps to understand which genes play a statistically significant role in certain biological processes in the human body.

The motivation behind this integrated analysis is to gain a comprehensive understanding of how NF-κB regulates gene expression across multiple biological domains. Advances in the study of NF-κB-related gene regulatory networks can help us pave better paths to fight these conditions. NF-κB is not limited to one biological process. It plays critical roles in inflammation, immune responses, cell survival, and stress signaling. By studying NF-κB, researchers can gain insights into mechanisms that affect multiple domains such as addiction (through neuroinflammation), cognition (via neural plasticity), and systemic homeostasis (through stress and metabolic regulation). We can elucidate the direct targets of NF-κB, map the complex interplay of regulatory interactions, and ultimately link these molecular insights to broader physiological and pathological outcomes. This holistic approach not only deepens our basic understanding of gene regulatory networks but also paves the path for innovative therapeutic strategies.[6]

One such study has been carried out below, where we relate the relevant genes with the NF-κB subunits and the biological processes they enrich:

Deriving the gene names interacting with each of the NF-κB subunits:

We will first find the genes interacting with each of the NF-κB subunits. We will then look for their influence on the biological processes later. The table below (Table 1) shows the sets of genes that were found interacting with each of the 5 subunits of NF-κB

Table 1: Genes that were found interacting with the specific subunits of NF-κB.

Genes interacting with **Nuclear factor NF-kappa-B p105 subunit:** ABCC2, CHUK, COPB2, CTNNB1, ESR1, HDAC1, HIF1AN, HTT, IKBKB, MAP3K8, MEN1, NFKB2, NFKBIA, NFKBIB, NOTCH1, PDCD11, PELP1, PLD3, RELA, RELB, RPS3, TNIP1, TNIP2, UL42
Genes interacting with **Nuclear factor NF-kappa-B p100 subunit:** MAP3K8, MEN1, NFKB1, NFKBIA, NFKBIB, REL, RELB, SUMO1
Genes interacting with **Transcription factor p65:** AATF, BANF1, BRD4, Brd4, CASP6, CCK, CDK5RAP2, CDK5RAP3, CHAT, CHUK, COMMD1, CREBBP, CRIP2, CTNNB1, DAXX, DHX9, EHMT1, ESR1, FGFR3, FUS, GAMMAHV.ORF73, GFI1, GSN, HDAC1, IER3, IKBKB, IRF5, KDM2A, KEAP1, KLF6, LAMP2, LMO2, MACROD1, MAPK10, MEN1, Myocd, NFE2L2, NFKB1, NFKBIA, NFKBIB, NFKBIE, nleC, NR4A1, NSD1, OGT, OTT_0753, OTT_1912, P/V, PDCD4, PIAS4, PPP2CA, PPP2R1A, PRPF40A, RAN, REL, RELB, RPS3, sctL, SETD7, SIRT1, SIRT2, SIRT6, SIX2, SNAI1, SOCS1, STAT3, TARDBP, TCF4, TGM2, TP53BP2, UBC, UBQLN1, UXT, UL42, yerA, YPO2940, YPO3877
Genes interacting with **Proto-oncogene c-Rel:** A1CF, ACOT12, ACOT8, AGPAT4, AIRIM, AKT2, ALOX5, AQP1, ARFIP2, ARIH2, ARL16, ARMC7, ASAP3, ASMTL, ATG9A, ATP6V0D2, ATP6V1C2, ATPAF2, ATXN1, BANP, BARHL2, BBS4, BCL2L15, BCL6, BID, BMF, C11orf68, C14orf119, C1orf50, C1orf74, C6orf142, C9orf72, CABP5, CACNA1S, CCNC, CCNJL, CDK18, CDKN1A, CDKN2C, CDKN2D, CDR2L, CENPX, CEP19, CFAP206, CGGBP1, CHAF1A, CHCHD2, CHIC2, CIB3, CINP, CKS1B, CLIP1, COL20A1, COL8A1, COMMD1, CPNE2, CPNE7, CPSF1, CSNK2A1, CTNNA3, CTNNBIP1, CYB5R2, DDX6, DEF6, DHPS, DMRT3, DNTT, DYNC1LI1, EFHC1, EGLN3, EHHADH, EIF3A, EIF3D, EIF4A3, EIF4E2, EIF4EBP1, EIF5A, EIF5A2, EMD, EML2, EPM2AIP1, ESRRA, ESRRG, EXOSC1, EXOSC5, EXOSC8, FAAP20, FAM200C, FAM90A1, FHL2, FKBP1B, FLAD1, FLNA, FNDC11, FNDC3B, FOXO4, FUT11, GADD45G, GLYCTK, GPKOW, GRAP, GRB2, HAT1, HDAC7, HIP1, HLA-DOA, HNRNPF, HSD17B14, HSPB7, IL36RN, KCTD6, KCTD7, KIAA0100, KLHL32, KLHL42, KRTAP9-4, L3MBTL2, LASP1, LENG1, LGALS14, LMO1, LMO2, LNPEP, LSM2, MAD2L2, MAGOHB, MAP3K7CL, MAPRE3, MB21D2, MBD3, MEMO1, MENT, MID2, MIEN1, MIPOL1, MMP2, MORF4L1, MORF4L2, MRPL10, MSRB3, MTURN, MYO15B, NABP1, NAGK, NCK2, NDUFAF3, NECAP1, NEDD9, NEIL2, NEUROG3, NFE2L2, NFKB1, NFKB2, NFKBIA, NFKBIB, NFKBIE, NGB, NME7, NMRK1, NOL9, NOXA1, NR2C2AP, NRSF, NUDT10, NUDT14, NXT2, OAZ3, OSGIN1, OSTF1, OTUB2, OTUD4, OTT_0753, OTT_1912, PADI3, PAK1IP1, PARVG, PATE1, PCYT2, PELI2, PGAP6, PICK1, PIH1D2, PLCB4, PLEKHN1, PM20D2, POLE2, POLR1A, POLR2L, POP5, PRDM10, PRKAA2, PRKAB2, PRPF31, PRPF40A, PSMA1, PSMA4, PSMB8, PTGER3, PTK6, QARS1, R3HDM2, RAB29, RAB41, RABIF, RAD23A, RAD51D, RASSF5, RBM39, RECK, RELA, RELB, REXO1L1P, RIPPLY1, RNF213, RNF6, RPS3, RSPH14, RTL8C, RUNX1T1, S100A1, SAMD11, SAT1, SCNM1, SDCBP, SEC14L4, SEC31A, SEPTIN7, SHC3, SIX1, SLC39A13, SLC41A3, SLC6A12, SMG9, SNRNP25, SOCS1, SPATC1L, SPG21, SPRY1, STAMBPL1, STAT3, STK16, SULT2B1, SUOX, SZT2, TBC1D21, TBC1D22B,
TCL1A, TCP11L1, THOC1, TLE5, TLX3, TNIP2, TNS2, TRAPPC2L, TRAPPC6A, TRIM68, TRIM74, TSC22D3, TSEN15, TSSC4, TSSK3, TTC19, TTC21A, TXNL4B, UBASH3A, UBASH3B, UBE2D4, UBE2K, UBE2Z, UBXN7, UBC, UXT, UL42, VPS25, WNK3, YARS, YJU2, YPO2940, YPO3877, ZBTB16, ZDHHC24, ZIC1, ZNF417, ZNF446, ZNF550, ZNF564, ZNF572, ZNF688, ZNF76, ZNF765
Genes interacting with **Transcription factor RelB:** COMMD1, GSK3B, NFKB1, NFKB2, RELA, SMARCC1

Deriving the biological processes enriched by each set of interacting genes:

We will now find the biological processes enriched by these genes, one set at a time. Only those biological processes are selected where the role of these genes has a higher significance (i.e., p-value < 0.05). This means that the influence of those genes on those biological processes is statistically significant, provided the sample size of the study done for those processes is high enough. Out of these biological processes, the top 10 processes are printed against the scale of -log10(p-value). This negative log (with base 10) keeps the values within the graph and helps with visualization since the real p-values are too far away in the linear scale. Here are the top 10 biological processes for each set of genes mentioned in Table 1. These are shown in Figures 1 through 5. Figure 1 below shows the top 10 biological processes that were influenced by the genes mentioned in Row 1 of Table 1 above. These are the genes that interact with subunit 1 of NF-κB.

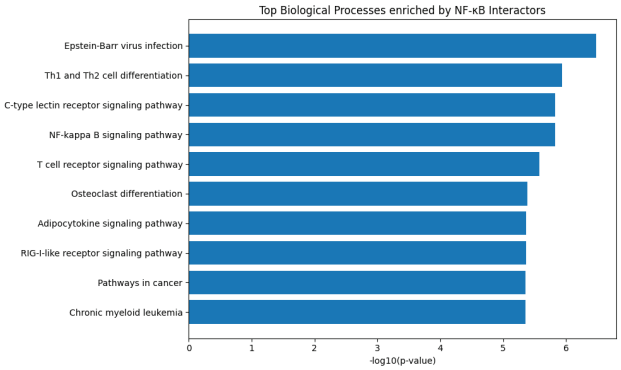

Figure 1: Top 10 biological processes enriched by genes that interact with subunit 1 (NF-κB B1-p50).

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Figure 2 below shows the top 10 biological processes that were influenced by the genes mentioned in Row 2 of Table 1 above. These are the genes that interact with subunit 2 of NF-κB.

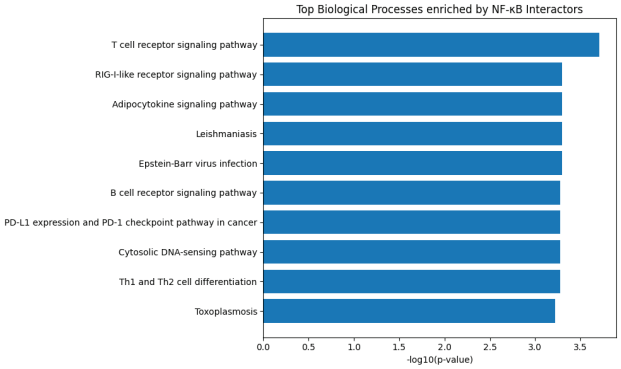

Figure 2: Top 10 biological processes enriched by genes that interact with subunit 2 (NF-κB2-p52).

Figure 3 below shows the top 10 biological processes that were influenced by the genes mentioned in Row 3 of Table 1 above. These are the genes that interact with subunit 3 of NF-κB.

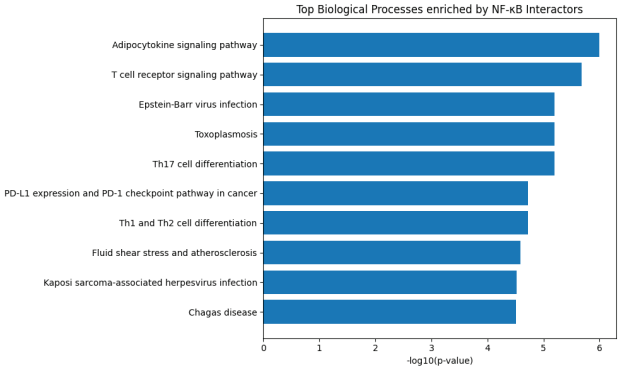

Figure 3: Top 10 biological processes enriched by genes that interact with subunit 3 (RelA-p65).

Figure 4 below shows the top 10 biological processes that were influenced by the genes mentioned in Row 4 of Table 1 above. These are the genes that interact with subunit 4 of NF-κB.

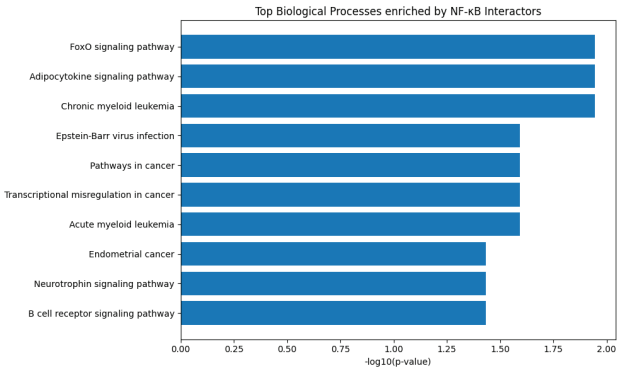

Figure 4: Top 10 biological processes enriched by genes that interact with subunit 4 (cRel).

Figure 5 below shows the top 10 biological processes that were influenced by the genes mentioned in Row 5 of Table 1 above. These are the genes that interact with subunit 5 of NF-κB.

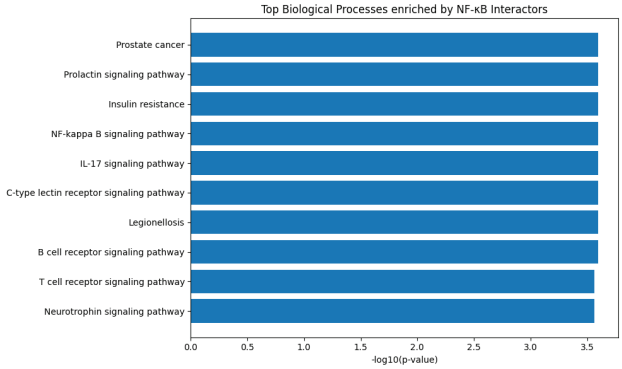

Figure 5: Top 10 biological processes enriched by genes that interact with subunit 5 (RelB).

Assessing gene commonality across the subunits:

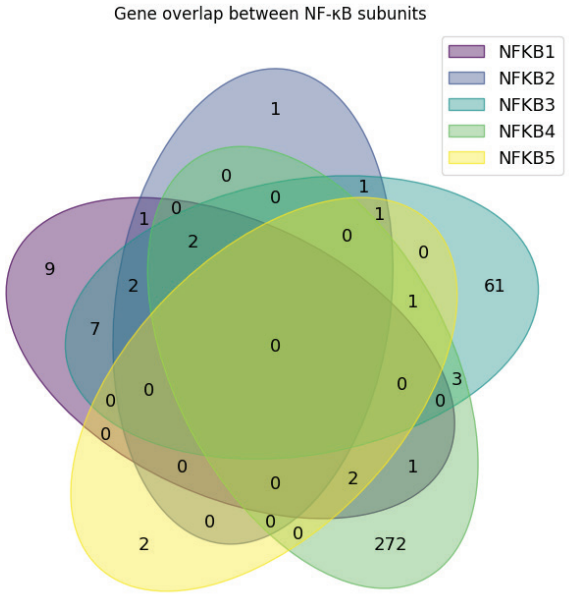

Figure 6: Assessing gene overlaps across the 5 subunits in NF-κB.

The Venn diagram in Figure 6 shows the overlap of the distinct genes interacting with each of the subunits of NF-κB. It is noteworthy that no single gene is common across all 5 NF-κB subunits. The data also goes on to show that each subunit is substantially exclusive and represents a diverse set of genes. Biological processes that are common to such a diverse set of genes can show promising relationships among the NF-κB subunits and genes influencing those.

Deriving common biological processes across the 5 sets of genes:

In this step, we will find the biological processes common across the 5 sets found above. Instead of taking just the top 10 biological processes, we take the entire record set of each of the 5 sets of biological processes formed above and carry out an intersection among them to find the common ones. The

function created for this purpose allows for choosing n number of records from each set of the biological processes and finding the overlap based on those. As an example, 100 records were chosen from each of the 5 record sets, and 9 biological processes were found to be common among them as of this writing. These are shown in Table 2 below. Note that this set can change if the program is run sometime in the future when the underlying APIs return different data.

Table 2: Biological process enhanced by all 5 sets of genes that interact with each of the NF-κB subunits.

Adipocytokine signaling pathway
Epstein-Barr virus infection
T cell receptor signaling pathway
Neurotrophin signaling pathway
Transcriptional misregulation in cancer
B cell receptor signaling pathway
PD-L1 expression and PD-1 checkpoint pathway in cancer
Chronic myeloid leukemia
Small cell lung cancer

Discussion

In this study, I have compared the attributes of the four genetic networks discussed above. The mesolimbic system helps the brain with dopamine levels and regulation. Correct amounts of dopamine at the correct times can increase motivation and learning to improve cognition. However, addiction can alter dopamine signaling, creating feelings of craving and the sensation to always want more.[7] Altered dopamine signaling makes it harder to balance stress and motivation, which can compromise neuronal homeostasis. The Acetylcholine network modulates neuronal excitability and how well the brain can react to stimuli.[9] The more connected the neurons are, the faster they can communicate. Faster communication creates faster reaction times and improves levels of cognition. Dopamine levels influence excitability levels of neurons, and repeated substance exposure affects the excitability in different ways, specific to the substance.[5] Maintaining neuronal excitability properly preserves neuronal homeostasis. The CREB-dependent network is important in coordination and communication between cells.[1] Faster communication within cells improves cognition and reaction time. Addiction can overstimulate reward pathways and can create tolerance from the repetition of substance abuse.[4] The network maintains neural stability. NF-κB pathways regulate neuroplasticity and play a crucial role in immune responses. They help neurons adapt to stimuli, which improves cognition. Addiction can create excessive dopamine release that can trigger chronic responses and neuroinflammation. They regulate stress and immune response.[6]

All 4 networks ultimately influence gene expression and contribute to various aspects of brain function, including reward processing, attention, memory, learning, and neuronal plasticity. As far as differences go, the mesolimbic system and the Acetylcholine network operate at the intercellular level, and their effects are often rapid and transient. CREB-dependent networks and NF-κB pathways are at the subcellular level, and they regulate gene expression for hours or even days.

We have found the biological process enhanced by the genes that interact with the NF-κB subunits. This finding can influence the direction in which those biological processes are being studied. For example, the biological process found to be common across the 5 NF-κB subunits suggests a stronger correlation of the entire NF-κB pathway with the life cycle of those biological processes. The experts studying NF-κB can provide valuable input into the research of these biological processes.

Further research should explore the temporal dynamics of gene expression through different addiction stages. Such research can help pinpoint the roles of each genetic network at every stage, which can be used for more specific and personalized treatments. For example, we can differentiate which processes create homeostatic responses and which are used in pathological states in severe addiction. Researching further into genetic polymorphisms and mutations can help personalize treatment as well.

Gene expression cannot always reflect actual protein use. For example, post-transcriptional regulation, protein modification, and degradation all affect protein quantities, and the functional significance remains as inferred.[6] The data used to determine NF-κB interactions are from network-based sources and do not include behavioral data.

This study supports that genetic networks are responsive to cognitive, addictive, and homeostatic demands, often bidirectionally.[6] The data analysis showed that genetic networks can influence a broad range of biological pathways throughout the body.

Conclusion

The Mesolimbic System and Acetylcholine Network interact closely in regulating reward, learning, and motivation.[7,9] Dopamine, the key neurotransmitter in the mesolimbic system, is influenced by acetylcholine, the key neurotransmitter in the acetylcholine network, which can enhance or inhibit dopamine release.[5,9] The NF-κB pathway is known for its role in immune response and interacts with the brain's neurotransmitter systems, usually under conditions of stress or neuroinflammation. This can disrupt dopamine signaling in the mesolimbic system, leading to depressive or cognitive-deficiency symptoms.[6] All four systems are connected because neurotransmitters (dopamine and acetylcholine) affect gene expression as seen in CREB and NF-κB pathways, which in turn change how neurons function over time.[1,6]

Deriving the genes interacting with each of the subunits of the NF-κB pathway and finding the common biological processes influenced by those genes shows that we should look at the NF-κB subunits in totality while also assessing those common biological processes. It also asserts the importance of considering the interplay between those genes while studying the proteins in the relevant NF-κB subunits and the biological processes influenced by them.

Looking forward, there is still much to be uncovered on genetic networks because of their quantity and specificity. For example, the total number of genetic networks or how many of them exist are questions that are still unanswered. We are

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also still unaware of the comprehensive locations of genetic networks and how far they span. Some factors of networks to research further include the longevity of networks, genetic variants in networks, the usage of these systems to create therapeutics for diseases, and the impact of external factors on them.

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