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Neurochemical Correlates of Captagon and Crystal Methamphetamine Addiction in Iraq: A Focus on Dopamine and Total Protein

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ABSTRACT

Background: Captagon (Kat) and crystal methamphetamine (crystal) are highly addictive stimulants that are widely used in Iraq. The neurochemical effects of these substances were examined in this study by comparing dopamine and total protein levels in users and a control group.

Methods: Blood samples were obtained from thirty control persons, fifty crystal addicts, and fifty Captagon addicts. The dopamine concentration was assessed utilizing an ELISA kit, whereas the determination of total protein levels was conducted using the Biuret method. Statistical analysis compared discrepancies between groups and the effects of age and body mass index (BMI).

Results and discussion: Dopamine levels were most significant among Crystal addicts, then those of Captagon addicts, and finally those of the control group. This indicates that dopamine release may have been elevated as a result of drug use. Crystal addicts had the lowest levels of total protein, which was considerably lower than the control group. This may be an indication of dietary deficits brought on by drug usage. The protein levels of captagon addicts did not differ significantly from those of the control group.

Variation in the impact of BMI on protein and dopamine levels was seen among the groups. In the lower BMI range (14-18.9), Crystal and control groups exhibited increased dopamine levels, whereas Captagon addicts exhibited comparable levels in both BMI ranges. Protein concentrations were marginally elevated within the lower BMI range in the control group. Age: Regardless of age, crystal users have consistently elevated dopamine levels (15-25 and 26-35). Within the younger age group, captagon addicts exhibited dopamine levels that were higher than the control group but lower than crystal users.

Conclusions: Addiction to Crystal and Captagon is correlated with unique modifications in levels of dopamine and protein. Crystal addicts demonstrated the most pronounced increase in dopamine levels and the lowest levels of protein, which may indicate neurochemical and nutritional alterations.

Keywords: *Dopamine, Protein, Captagon, Crystal Methamphetamine*

1. INTRODUCTION

Substance dependence is a chronic relapsing condition that is distinguished by the following symptoms: an inability to restrain one's intake, a negative affective state (e.g., dysphoria, anxiety, irritability), and an irresistible desire to get and use the substance. The diagnostic classification of substance use disorders now encompasses the term addiction.[1]. Substance addiction is a multifaceted condition that is heavily influenced by genetic factors. Morphine and heroin exert their effects via the mu-opioid receptor (OPRM1), which is the principal target of opioid medicines and peptides. OPRM1 also influences substance abuse addiction through its impact on dopaminergic pathways, which include cocaine, nicotine, and alcohol.[2] . Substance abuse is a chronic biochemical illness that impacts both the brain and behavior of individuals, resulting in compulsive drug use. Complex alterations in reward, motivation, neuroplasticity, memory, and cognitive control are encompassed within this phenomenon, alongside other interconnected cerebral circuits and regions. [3] Neuroimaging research has successfully identified distinct cerebral areas that are implicated in various phases of addiction.

In the binge/ intoxication stage, the ventral tegmental area, nucleus accumbens, and caudate nucleus are involved; in the withdrawal/negative affect stage, the orbitofrontal cortex, dorsolateral prefrontal cortex, amygdala, and hypothalamus are involved. [4] A dysregulation of the insula and prefrontal lobes occurs during the anticipation stage, which results in the development of cravings. [5] Addiction is an illness characterized by chronic relapse; hence, knowledge of its neurobiology can facilitate the creation of productive treatment approaches. [6] DSM-51 merged substance abuse and substance dependence, which were previously considered distinct and hierarchical disorders, into a single construct in 2013. Substance use disorders were categorized along a continuum ranging from mild to severe, with the extent of an addiction being determined by the number of established criteria that are met. A primary objective of neurobiological research is to comprehend the molecular, cellular, and neurocircuitry-level alterations that facilitate the progression from infrequent, regulated substance use to chronic addiction characterized by uncontrolled drug consumption.[7]. There has been a growing interest in neurobiological mechanisms that influence the different individual differences in drug responses, as only a subset of substance users successfully transition. There have been persuasive arguments positing that addictions bear a resemblance to other chronic relapsing illnesses, including hypertension, diabetes, and asthma, in which individual reactions to identical external stressors vary, and treatment efficacy is restricted.[8]. Addiction may be defined as a recurring cycle comprising three stages: binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation (craving). This cycle progresses in severity and is characterized by Brain regions involved in reward, stress, and executive function undergoing neuroplastic changes [9]. Constructed upon a convergence of insights from psychiatry, brain imaging, and social psychology about human self-regulation failure, these three phases offer a heuristic framework for investigating the neurobiology of addiction.[10]. Impulsivity is "a propensity for swift, unplanned responses to internal and external stimuli, irrespective of the adverse repercussions these responses may have on oneself or others."[11]". Compulsivity is characterized by the outward display of "persistent, repetitive behaviors that are both excessive and improper in nature." While impulsive actions are frequently motivated by pleasure or gratification, compulsions manifest in conditions like obsessive-compulsive disorder as a means of alleviating anxiety or tension caused by preoccupied thinking. Within this particular framework, people progress from impulsivity to compulsivity, and the impetus for engaging in drug-related activities coincides with changes in reinforcement from positive to negative. Compulsivity and impulsivity can, however, coexist and do so frequently throughout the many stages of the addiction cycle.[12]. In addition, they can cause physical or mental dependence and addiction, with stimulants, including amphetamines, being the most detrimental.[13] Methamphetamine ranks second in terms of frequency of illicit substance usage, following cannabis. They harm health when consumed internationally, particularly in East and South-East Asia. In the previous year, 37 million individuals took amphetamines, according to the UNODC, with MA being the most widely used substance in China. Synthetic drug users outnumber heroin users. After 2016, 60.5% of all drug users were artificial drug users[14] . Prominent specimens of central nervous system stimulants that augment dopamine secretion in the brain are cathinone (methylenedioxy-methamphetamine) and Methamphetamine (MAMP). As a stimulant of the central nervous system, methamphetamine produces euphoria, hallucinations, and heightened wakefulness. Substances possessing significant reinforcing and addictive properties are susceptible to tolerance and abuse, culminating in the ingestion of toxic quantities by users.[15]

Phenethylamine, or captagon, is a chemical that has a resemblance to amphetamine Its chemical formula is (18H23N5O2C)[16]. It induces sleep problems, increased activity, excessive movement, absence of appetite, and exhaustion and is classified as a stimulant. Cathinones are classified as illegal synthetic substances. They are produced in Eastern European nations such as Bulgaria and Poland before making their way to Syria and Turkey, from where they are trafficked via Jordan and Iraq to Saudi Arabia and the Gulf states.

Substance misuse drugs stimulate reward systems in the brain, and the neurocircuitry of reward has been substantially defined by research on drug addiction. The significance of this line of inquiry lies in the fact that alterations in the activation of the drug-induced reward system are crucial to comprehend the progression of addiction.[9]. In this context, "reward" refers to any occurrence that heightens the likelihood of a reaction, including a positive hedonic component. Extensive investigation into the neurobiology underlying the rewarding effects of abused substances has centered on the terminal regions and origins of the ascending mesocorticostriatal dopamine systems, which are crucial in regulating the rewarding characteristics of most such substances. [17]. Positron emission tomography studies have demonstrated that intoxication amounts of drugs and alcohol cause the ventral striatum to produce dopamine and opioid peptides in humans. [18] indicating that rapid and abrupt dopamine release[19]is correlated with the individual perception of the so-called "high."[20]. This is because rapid and substantial surges in dopamine stimulate low-affinity dopamine D1 receptors, essential for the pleasant pharmacological effects. [19]as well as to induce conditioned reactions. 38 Dopamine activation of high-affinity dopamine D2 receptors, in contrast, does not confer adequate incentive for drug use. [21] Moreover, these receptors may restrict drug reward.[22]. Drugs mimic the dopamine surge initiated by phasic dopamine binding, which refers to the firing frequency of dopamine neurons linked to pleasurable stimuli.

[23]. The dopaminergic system plays critical role in mediating behavior effects and artificial reward with brain stimulation reward and addictive drugs[24]

This research endeavor aims to determine the consequences of crystal and cathinone use on the dopamine system within the body by analyzing protein and dopamine concentrations in users. By quantifying protein and dopamine levels, one can acquire a more comprehensive comprehension of the chemical alterations that transpire within the organism due to the use of drugs.

2. MATERIALS AND METHODS

Thirteen samples, aged between 15 and 35, were collected from substance users and control groups at Al-Ataa Hospitals for Addiction Treatment and Psychological Rehabilitation, the Department of Forensic Medicine, and Ibn Rushd Teaching Psychiatric Hospital in Baghdad, Iraq, for the current study. The research spanned the months of February 2022 through September 2023.

The samples were divided as follows:

Group 1: This group included 50 people with an addiction to crystal methamphetamine. Group 2: This group included 50 people with an addiction to khat (**Captagon**).

Group 3: This group included 30 control samples.

Five milliliters of venous blood were collected from the substance users and placed in clot-free tubes for biochemical analysis. Based on the Biuret Method, the serum total protein concentration was determined with a commercially available kit and the colorimetric method

outlined in the kit.[25] . The dopamine concentration was measured by following the instructions provided with the specific ELISA analysis kit, coded Rust) ER0500 and manufactured by Faulhaber (2006)[26].

Body mass index is a simple weight index for height ordinarily used to distinguish insufficient weight, average, overweight, and obesity. It is calculated by dividing the weight in kilograms on the height square in meters (kg/m2) presenting to the following equation[27]:-

BMI = Weight (Kg) /(Height)2 (m^2)

3. STATISTICAL ANALYSIS

Data were analyzed by the Minitab program system ver., 17. and the ANOVA tests were applied. The means were compared by Duncan's Multiple Range Test (DMRT) under the level of significantly 0.05

4. RESULTS AND DISCUSSION

A distinct disparity in dopamine concentrations among the three groups is seen in Table 1 and Figure 1. Specifically, Crystal addicts (G1) demonstrate the highest mean dopamine levels at

679.00 pmol/ml. This value is considerably more than that of the control group and captagon addicts (G2) (G3). Captagon addicts (G2) exhibit a moderate increase in dopamine levels (mean: 437.40 pmol/ml) in comparison to the control group. Conversely, the control group (mean: 124.39 pmol/ml) has the lowest dopamine levels. Enhanced Dopamine Secretion, It is known that both crystal meth and captagon function by boosting the release of dopamine in the brain. This may explain the higher dopamine levels found in groups G1 and G2 compared to group G3. The heightened dopamine secretion is accountable for the gratifying benefits correlated with these substances; nevertheless, it may also foster addiction and give rise to further adverse outcomes.[28, 29]. Dopamine (DOP) is a chemical messenger that, when produced by other nerve cells, sends signals to nerve cells. Dopamine's pathways in the brain regulate the production of numerous hormones [30]. Sensitivity or Dopamine Receptor On the contrary, variations in dopamine levels among people with an addiction may be attributable to modifications in the sensitivity of dopamine receptors in their brains. Chronic drug use has the potential to induce downregulation of dopamine receptors, resulting in a diminished effect of an equivalent quantity of dopamine. This may account for the possibility that G2, although possessing lower dopamine levels than G1, could exhibit comparable addictive behaviors [31, 32].

An important role is played by dopamine in addiction. The majority of addictive medications increase extracellular dopamine levels, which can influence the formation of long-term memories that link predictive inputs with rewards and punishments and ascertain the incentive to react to such signals, according to research.[33] Additionally, methamphetamine and similar substances are psychomotor stimulants. They increase levels of dopamine.[34] In addition, exercise intervention can lessen drug dependence by enhancing dopamine levels, immunity, and the mitigation of unpleasant emotions, according to a study on amphetamine addiction.

[35] .Moreover, there has been speculation regarding a possible correlation between the severity of gambling addiction and levels of central dopamine. This suggests that illnesses involving the dysregulation of striatal dopamine transmission could be linked to addiction disorders.[36]

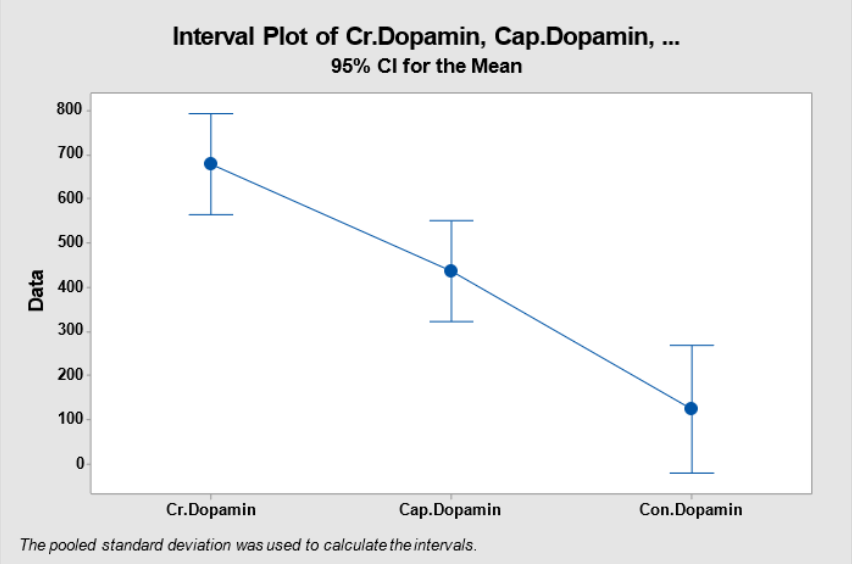

Figure 1. Dopamine Levels in the study groups

A distinction in protein levels among the three groups is illustrated in Figure 2 and Table 1: The protein levels of crystal addicts (G1) are the lowest, averaging 5.546 g/dL. This is considerably less than that of the control group (G3). Captagon addicts (G2) exhibit comparable protein levels as crystal users, averaging 5,6600 g/dL. Statistically speaking, this difference is insignificant compared to the control group. The protein concentration in the control group (G3) is the

highest, averaging 7.2290 g/dL. Condition of Nutrition: Hypoglycemia may be suggested by reduced protein levels in groups G1 and G2, which may be attributed to the adverse impacts of medication on appetite and dietary absorption.[37]. Chronic drug use has the potential to induce inflammation within the body, resulting in a reduction in protein synthesis and subsequent decline in protein levels.[38]. Hepatic function: The primary site of protein metabolism is the liver. Substance abuse-induced liver damage may impede protein metabolism and contribute to decreased levels.[39]. Mass of Muscle: Protein is an essential component of muscular tissue. Protein levels in G1 and G2 may be influenced by a reduction in muscle mass resulting from medication use.[40]. Amino acids form the cornerstone of the construction of proteins and peptides and play a key role as a means of food construction[41]

In contrast, a research investigation examining the impact of an eight-week interval training program on the expression of the amyloid precursor protein (APP) gene in the hippocampal tissue of rats dependent on methamphetamine revealed the following: Methamphetamine significantly increased APP gene expression in comparison to the control group, whereas the training program resulted in a substantial decrease in APP expression in contrast to the groups that received methamphetamine alone.[42]. An instance of acute nephrotoxicity resulting from Captagon was documented in a study when a patient exhibited a total protein level of 3.2g/dL, which falls below the established normal range of 5.0–9.0g/dL.[43]. Alcohol abuse and other lifestyle-related factors commonly observed in individuals with mental disorders have been linked to changes in hepatic cytochrome P450 (CYP) protein levels. These modifications can potentially impact drug metabolism and the effectiveness of frequently prescribed medications. [44]

The study's Shalash, W.K. and Z.H. Kadri findings demonstrated a strong correlation between cytotoxicity and serum levels of MDA and C-reactive protein. The strength of this correlation varies according to a person's genetic predisposition, the number of cigarettes or narghiles they smoke each day, the length of time they are exposed to toxins, and the gases they release[45].The environment is constantly contaminated by heavy metals, some of which are needed to sustain metabolism and activity, others of which have no bearing on biology, and the concentration of these metals when they rise causes toxicity to living organisms, including the basic body mineral lead from toxic and non-essential metals even in low doses that affect the nervous system, blood, genitalia, and body's cognitive function as well as IQ[46]. When intake of

Cu exceeds the range of biological tolerance, it can cause adverse effects, including damage to liver, kidney, immune system, and gastrointestinal distress[47]

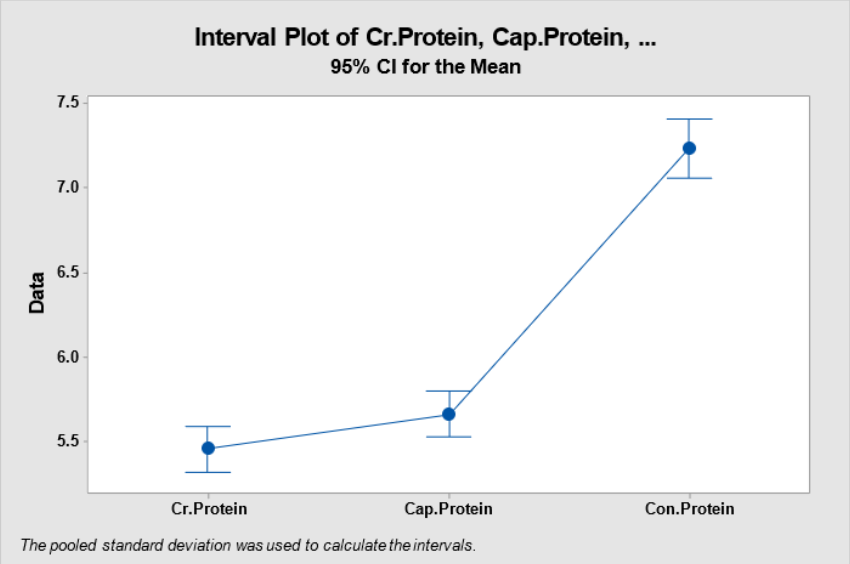

Figure 2. Protein Levels in the study groups Table (1): Parameter Levels in the study groups

Table (1): Parameter Levels in the study groups

Parameter	**Groups**	**Means ± SD**
Dopamin (pmol/ml)	G1 (n=50)	679.00 ± 65.90 a
	G2 (n=50)	437.40±59.30 b
	G3 (n=30)	124.39±55.22 c
Protein (g/dL)	G1 (n=50)	5.546 ± 0.584 b
	G2 (n=50)	5.6600±0.2976 b
	G3 (n=30)	7.2290±0.553 a

G1: Crystal addict , G2: Captagon addict, G3: Control

The dopamine levels in three BMI ranges for three distinct groups (Crystal addict, Captagon addict, and control) are presented in TABLE 2 and Fig 3. The dopamine levels within each group can be compared between the two BMI ranges to determine how BMI influences these outcomes. Dopamine levels in G1 (Crystal addicts) are more significant in the range of 14 to 18.9 BMI than in the range of 19 and above BMI. The dopamine concentration in G2 (a cocaine addict) is comparable across the two BMI ranges. Dopamine levels are similarly more significant in the 14-18.9 BMI range in G3 (Control) than in the 19 and above BMI range. As a result, the influence of BMI on dopamine levels differs between groups, with the Crystal addict and control groups exhibiting higher dopamine levels in the lower BMI range.

The effect of drug abuse on BMI in individuals is a complex issue influenced by various factors. Research has shown that prenatal drug exposure, such as polytobacco/opioid use, can lead to lower pediatric BMI percentiles in children from 2 to 16

years of age[48]. Across all demographics, persons with higher BMIs have lower dopamine levels. This may result from several factors: the food-dopamine relationship, Obesity-induced alterations in reward circuits, and hunger management may modify the dopamine response to the medications.

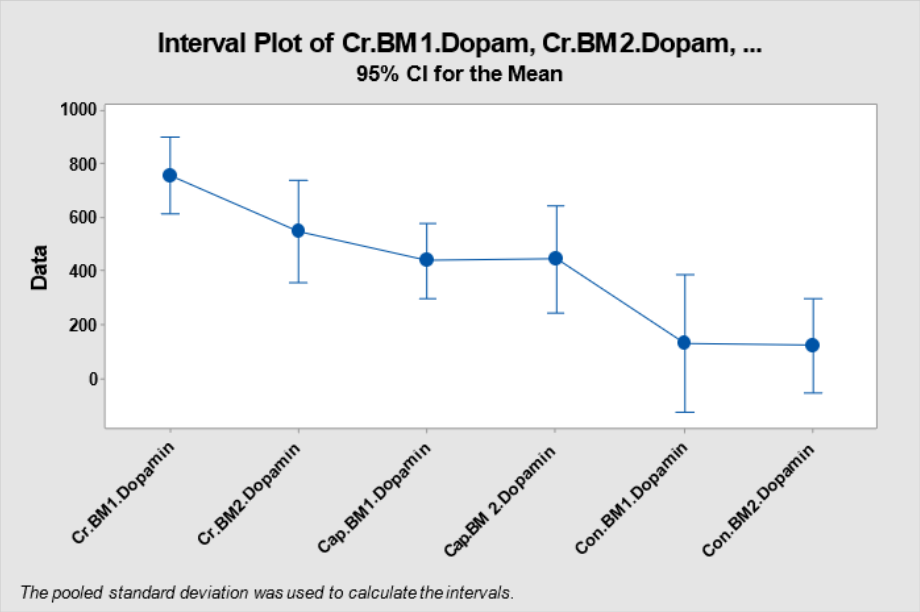

Figure 3. The effect of BMI on Dopamine in the study groups

Furthermore, a study on HIV-positive drug abusers revealed that cocaine abuse was associated with lower BMI, while strict opiate abuse was not[49]. The protein concentrations of three distinct groups (Crystal addict, Captagon addict, and control) for two BMI ranges are displayed in TABLE 2 and Fig 4. Protein levels in the G1 (Crystal addict) and G2 (Captagon addict) groups are comparable throughout both BMI ranges. No significant variation in protein levels can be observed among these groups according to BMI. In the G3 (Control) group, protein levels differ significantly according to BMI. The average protein concentration is considerably greater (7.090 g/dL) in the BMI range of 14-18.9 than in the BMI range of 19 and older (7.295 g/dL). Potentially attributable to the low protein content of users G1 and G2. Substance abuse can cause nutritional disturbances and insufficient protein consumption, which in turn contributes to diminished levels.[50].

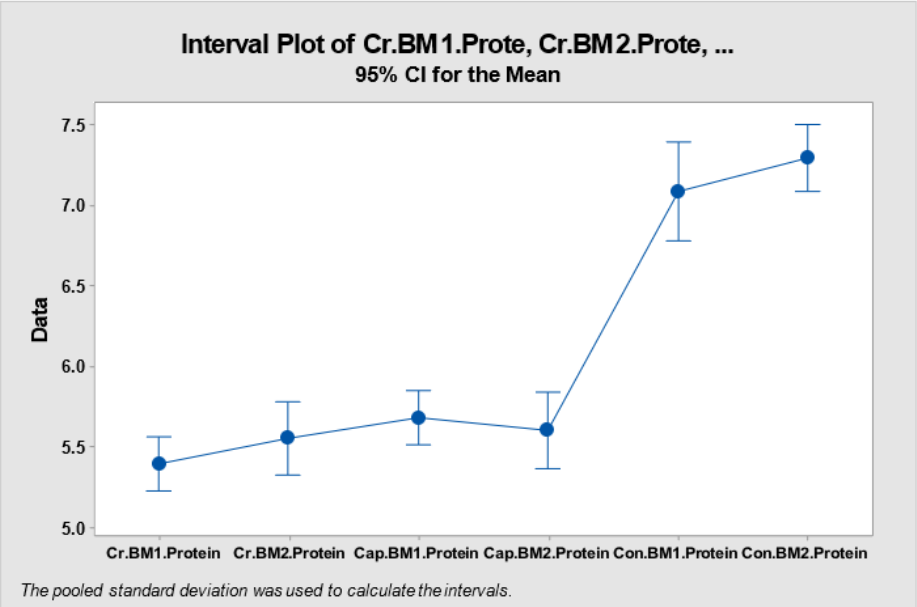

Figure 4. The effect of BMI on Protein in the study groups

Table (2): The effect of BMI on Parameter Levels in the study groups

Groups	B M I	Parameter	
		Dopamine (pmol/ml)	Protein (g/dL)
G1	14 – 18.9	755.0 ± 73.1 a	5.397 ± 0.629 b
	19 – Above	544.7 ± 67.9 b	5.561 ± 0.495 b
G2	14 – 18.9	435.5 ± 43.4 b	5.685 ± 0.265 b
	19 – Above	441.6 ± 56.9 b	5.606 ± 0.360 b
G3	14 – 18.9	128.7 ± 22.0 c	7.090 ± 0.433 a
	19 – Above	122.3 ± 23.2 c	7.295 ± 0.600 a
P-Value		0.01 **	0.01 **

G1: Crystal addict , G2: Captagon addict, G3: Control

The findings are presented in Figure 4 and Table 3, which illustrate the impact of age on dopamine levels across three distinct groups: control, crystal addicts (G1), and captagon addicts (G2) (G3). Dopamine concentrations were assessed in two age cohorts: 15–25 and 26–35. According to the data, G1 had the most excellent dopamine levels in both age groups, followed by G2 and G3. G1 had considerably elevated dopamine levels compared to G2 and G3 in the 15-25 age group. Similarly, in the 26-35 age group, G1 had significantly elevated dopamine levels compared to G2, and G2 had significantly elevated dopamine levels compared to G3. Age may influence dopamine levels, and addiction to crystal or Captagon may also have an effect, according to these findings. Greater Dopamine Levels in the Older Age Group: For all three age groups, the data demonstrates a clear tendency of greater dopamine levels in the 26-35 age group compared to the 15-25 age group (G1, G2, and G3). This may result from the following factors, among others: The process of dopamine system maturation[51]; dopamine system development persists into adolescence and early adulthood, culminating in its maximum functionality at approximately 30 years of age. This may account for the elevated dopamine concentrations reported in older people. In life experiences[45], dopamine involves many reward-related mechanisms, encompassing learning, memory, and motivation. Individuals aged 26 to 35 may have amassed a more significant number of life experiences that have influenced their dopamine system's development and activity levels. Substance abuse is a critical public health issue that results in substantial morbidity and mortality, particularly among adolescents and populations at the highest risk.[52] Alterations in brain anatomy, including a reduction in the density of dopamine transporters, which may be associated with aging, may account for the observed variations in dopamine concentrations.[53].

Across both age groups, crystal addicts (G1) consistently exhibit the most significant amounts of

dopamine. This is consistent with dopamine's recognized involvement in addiction and reward- seeking behavior. In the younger age group, captagon addicts (G2) have reduced dopamine levels in comparison to G1 but greater levels than the control group (G3). This implies that the impact of these two medicines on dopamine systems may differ. Younger age group captagon addicts (G2) have higher dopamine levels than the control group (G3) but lower levels than G1 addicts (G2). This observation implies the possibility of a distinction in how these two categories of medications impact dopamine systems.

Table (3): The Effect of Age on Parameter Levels in the Study Groups

Groups	Age/years	Parameter	
		Dopamine (pmol/ml)	Protein (g/dL)
G1	15 - 25	596.9 ± 57.6 b	5.574 ± 0.584 b
	26 - 35	813.0 ± 51.3 a	5.263 ± 0.604 b
G2	15 - 25	446.8 ± 46.7 c	5.612 ± 0.332 b
	26 - 35	427.3 ± 59.9 c	5.713 ± 0.250 b

G3	15 - 25	140.6 ± 55.9 d	7.276 ± 0.533 a
	26 - 35	90.3 ± 36.4 d	7.130 ± 0.609 a
P-Value		0.01 **	0.01 **

G1: Crystal addict , G2: Captagon addict, G3: Control

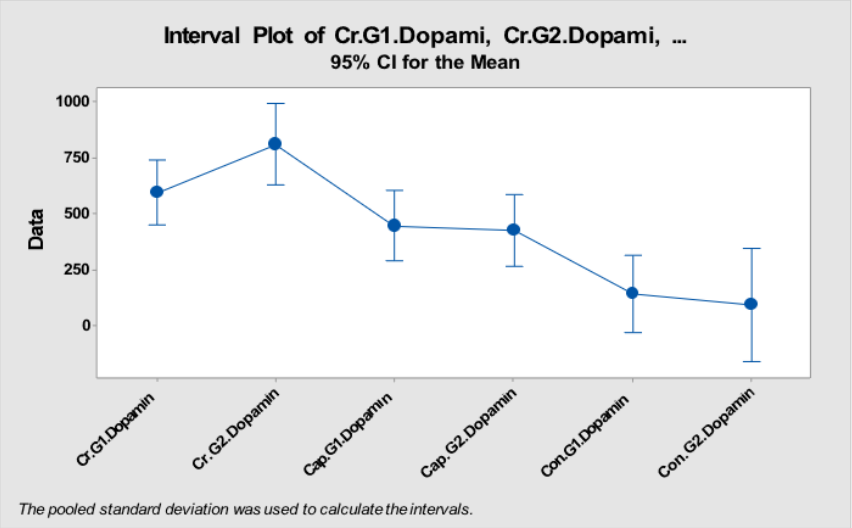

Figure 5. The effect of Age on Dopamine in the study groups

Table 3 and Figure 6 indicate the protein levels. The control group (G3) consistently exhibited the highest protein quantities in both age groups. Protein concentrations were reduced in both medication groups (G1 and G2) compared to the control. The age effect shows that protein levels decline across all age groups. The reduction in the control group is marginal (7.28 to 7.13), whereas the decline in both drug groups is significantly more pronounced (5.61 to 5.71 for G2 and 5.57 to 5.26 for G1). Age-related decline in protein metabolism[54]. Protein breakdown (catabolism) may increase when protein synthesis (production) declines with age. This may result in decreased protein levels overall. The presence of nutritional deficits and inadequate dietary practices may be linked to substance misuse, perhaps resulting in nutritional inadequacies that hurt protein levels.[55]. Increased catabolism of proteins; specific compounds, such as Crystal and Captagon, have the potential to induce protein degradation, resulting in reduced protein levels.[56]. Inflammation: Chronic inflammation caused by substance usage may also contribute to elevated protein degradation and decreased levels.[57].

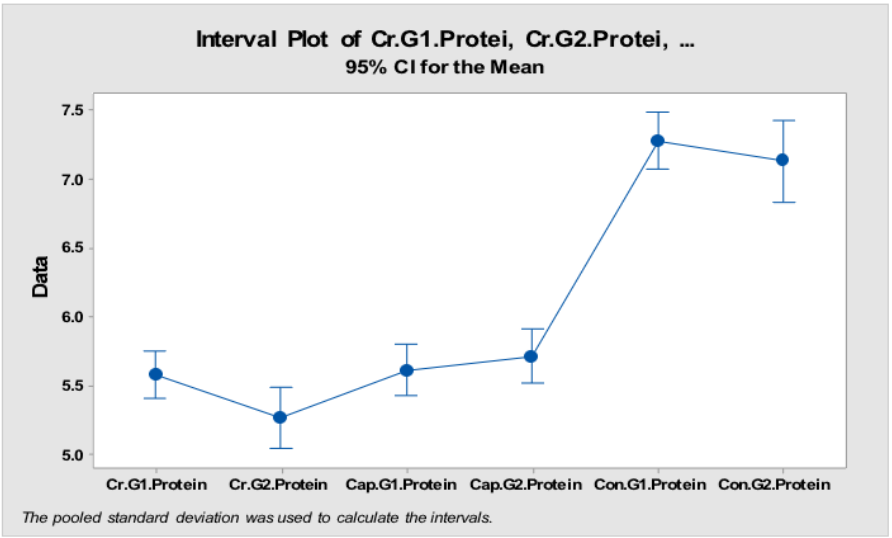

Figure 6. The effect of Age on Protein in the study groups

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