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# PLATELET MAO ACTIVITY IN CHRONIC SCHIZOPHRENIA

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SUMMARY

Platelet MAO activity of 30 male drug free chronic schizophrenics (RDC), 30 male first degree relative of these patients and 31 age matched male healthy controls was studied. Analysis of variance revealed that no significant differences existed between the three groups. There was high correlation between the enzyme activily of pco-bands and first degree relatives. Presence or absence of auditory hallucinations, or paranoid features did not affect the enzyme activity significantly.

## Introduction

Monoamine oxidase (MAO), an insoluble mitochondrial enzyme, metabolises a number of biologically active monoamines most of which are known neurotransmitters or neurotransmitter candidates. It has been implicated in the causation of various neuropsychiatric illnesses. A positive correlation between brain and platelet MAO activity has been observed by Robinson and associates in 1971. Easier accessibility to platelet MAO as compared to brain MAO has resulted in a number of studies of platelet MAO activity in order to explain pathophysiology of various neuropsychiatric illnesses. Murphy and Wyatt (1972) have observed a decreased platelet MAO activity in chronic schizophrenic psychosis and suggested that platelet MAO activity can serve as a biological marker for schizophrenia. Since then 42 studies have been reported in world literature. 26 of these have replicated the above findings, while others have not, issue is still controversial. In order to study this important biological variable in Indian patients, we carried out this work with the following aims:

1. To study platelet monoamine oxidase (MAO) activity in a group of chronic schizophrenia and matched controls.
2. To study platelet MAO activity in first degree relatives of patients of chronic schizophrenia.

## Material and Methods

A. Patient Sample: 30 patients suffering from chronic schizophrenic psychosis were included in the study according to following inclusion and exclusion creiteria –

*Inclusion Criteria* –

(a) RDC diagnosis of chronic schizophrenic psychosis (Spitzer *et al* 1978).

(b) 17 to 60 years of age.

(c) Male sex

(d) Resident of Lucknow

*Exclusion Criteria* –

(a) History of neuroleptic intake within past one week

(b) Presence of anaemia (Hb < 10Gm %)

(c) Evidence of organic brain syndrome

(d) History of alcohol abuse or any other drug abuse (e.g. cannabis, marijuhana, opium) within past 6 months.

(e) Non-availability of required male first degree relative.

(f) Absence of consent

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B. Control : 31 age matched male healthy volunteers were selected as controls from amongst the non-professional hospital staff and relatives of surgical patients. Following exclusion criteria were strictly observed –

(a) Hb <10 Gm %

(b) History of Neuro-psychiatric illness (migraine, Huntington's chorea, schizophrenic psychosis, affective disorder) in the subject himself or his first degree relatives.

(c) Presence of physical illness requiring medication.

(d) History of alcohol abuse or any other drug addiction (Cannabis, marijuana, opium) within past 6 months.

(e) First degree relatives : One male first degree relatives for each selected by random sampling was taken up for estimation of platelet MAO activity.

All patients attending psychiatry outpatients department at G.M. and Associated Hospitals, Lucknow were screened for inclusion in the study. First 30 patients fulfilling the inclusion–exclusion criteria were hospitalised and kept drug free. They were examined independently by two psychiatrists to ensure the diagnosis. First degree relative included in the study was also examined for the presence of any physical or psychiatric illness. All data were recorded on a semi-structured proforma. Blood samples were collected on the day of hospitalisation in the morning between 10.00 A.M. and 12.00 noon after the clinical examination was completed.

17ml. of venous blood was collected through vene-puncture into plastic syringes (steriware) rinsed with acid citrate dextrose solution, and was then transferred immediately into two polypropylene tubes each containing 1.5 ml. of acid citrate dextrose (ACD) solution. The tubes were immidiately placed in ice. Platelet rich plasma (PRP) was prepared within 4 hours by differential refrigerated centrifugation at 4°C. Centrifugations were done at 175g., 300g. and 600g. for 10 minutes each and PRP obtained after each centrifugation was pooled (Sullivan *et al* 1977). Platelet counts were done in PRP using light microscope and Neubauer's chamber. PRP was stored at – 70°C till the time of assay, but never more than 2 weeks (No sufficient change in enzyme activity occurs at this temperature for 6 weeks, Murphy *et al* 1976). At the time of assay PRP was thawed. Platelet pellets were obtained by centrifuging PRP at 3000g. for 10 minutes in a refrigerated centrifuge. Pellets were washed with normal saline and then sonicated with 0.1 ml distilled water for each ml of PRP and were separated into alliquots of 0.2 ml. Platelet MAO activity was estimated in these alliquots using benzylamine as substrate by spectro-photo-meteric method of Tabor *et al* (1955). Proteins were estimated in these alliquots by method of Lowry *et al* (1951). Estimaters were blind to the identity of the sample. All the estimations were done at Industrial Toxicology Research Centre. All the samples were run in duplicate. Samples from patients and corresponding first degree relatives and controls were obtained on the same day and their estimations done the same day. Enzyme activity was calculated in terms of *n* moles of benzaldehyde formed per $10^8$ platelets per hour and also *n* moles of benzaldehyde formed per mg of platelet protein per hour. Statistical analysis was done using one way analysis of variance.

## Results

*Sample Characteristics :*

We studied 30 male patients of chronic schizophrenia (RDC) with an average age of 30.03 (±6.86) years, age of onset of illness 23.03 (±7.53) years and average duration of illness 7.17 (±4.43) years (Table 1). Controls were 31 males with an average

**Table 1**
**Sample Characteristics**

| | Patients (N = 30) | Ist Relatives (N = 30) | Controls (N = 30) |
|---|---|---|---|
| **AGE (in years)** | | | |
| Mean | 30.03 | 41.43 | 30.39 |
| S.D. | 6.86 | 18.57 | 7.39 |
| Range | 19-45 | 19-71 | 18-46 |
| **Hb (Gm %)** | | | |
| Mean | 11.45 | 11.57 | 11.72 |
| S.D. | 1.01 | 0.99 | 1.29 |
| Range | 10.0-14.0 | 10.0-14.0 | 10.0-14.5 |
| **Platelet Count** x $10^8$/cc of PRP | | | |
| Mean | 2.54 | 3.06 | 2.81 |
| S.D. | 1.10 | 1.69 | 1.55 |
| Range | 1.0-4.26 | 1.11-8.41 | 0.88-5.77 |

age 30.39 (± 7.30) years. First degree relatives were 30 males with an average age 41.43 (±18.57) years with a range of 19 to 71 years, 3 of them were sons, 12 were fathers and 15 were brothers. The mean haemoglobin was 11.45 gm % for first degree relatives. Platelet counts were $2.54 \times 10^8$ per cc of PRP for patients, $3.06 \times 10^8$ per cc of PRP for first degree relatives and 2.81 x $10^8$ per cc PRP for controls. We were unable to observe any enzyme activity in platelets of 4 patients, 4 first degree relatives and one control because their enzyme activity was so low that it could not be detected by our method of estimation. These subjects were excluded from further statistical analysis.

**Table 2**
**Platelet MAO Activity in Patients, First Degree Relatives and Controls**

| | Patients (N = 26) | Ist Relatives (N = 26) | Controls (N = 30) |
|---|---|---|---|
| n Moles/$10^8$ Platelets/hr. | | | |
| Mean | 24.15 | 24.77 | 30.25 |
| S.D. | 25.07 | 21.27 | 28.56 |
| | F 2.81 = 0.003, N.S. | | |
| n Moles/mg Platelets protein/hr. | | | |
| Mean | 43.78 | 60.82 | 59.62 |
| S.D. | 43.28 | 63.75 | 69.95 |
| | F 2.79 = 0.45, N.S. | | |

The platelet MAO activity did not differ significantly between the three groups i.e. patients did not differ from controls or first degree relatives, and first degree relatives did not differ from controls.

**Table 3**
**Correlation Coefficient Between Platelet MAO Activity and other Variables**

1. Correlating between two values of platelet MAO activity (r) = 0.81, (p<.001)
2. Correlation between platelet MAO activity of patients and Ist relatives (r) = 0.56, (p<.01)
3. Correlation between age of onset of illness and platelet MAO activity (r) = −0.14 (NS.)
4. Correlation between duration of illness and platelet MAO activity (r) = −0.17 (N.S.)

There is a highly significant correlation (r = 0.81, p<.001) between two values of platelet MAO activity for the whole group. There was a significant correlation (r = 0.56, p<.01) between the platelet MAO activity of patients and their first degree relatives. However no correlation was seen between platlet MAO activity and either the age of onset of illness or the duration of illness.

**Table 4**
**Comparison beween treated and untreated patients and MAO values**

| | Treated Patients (N = 5) | Untreated Patients (N = 21) | Control (N = 30) |
|---|---|---|---|
| n Moles/$10^8$ Platelets/hr. | | | |
| Mean | 9.17 | 27.72 | 30.25 |
| S.D. | 12.05 | 26.03 | 28.56 |
| | F 2.55 = 1.28, N.S. | | |
| n Moles/mg. platelet protein/hr. | | | |
| Mean | 10.59 | 51.71 | 59.62 |
| S.D. | 6.75 | 44.51 | 69.95 |
| | F 2.53 = 1.45, N.S. | | |

In order to exclude the effect of neuro leptic treatment on platelet MAO activity the sample was divided into two groups. There were only 5 patients who presented with a history of neuroleptic treatment for a variable period of two months to ten years in the past. Three of them were drug free

for eight days, one for twenty five days and one for one and a half years prior to inclusion in the study. Twenty one patients had not received any treatment with neuroleptic drugs in the past. Treated groups of patients tended to have lower values of platelet MAO activity as compared to untreated group of patients and controls. But this difference failed to reach any statistical significance.

**Table 5**
**ANOVA of platelet MAO activity according to presence or absence of hallucinations**

| | Patients with Auditory hallucinations (N=17) | Patients without Auditory hallucinations (N = 9) | Control (N = 30) |
|---|---|---|---|
| n moles/$10^8$ Platelets/hr | | | |
| Mean | 26.42 | 21.65 | 30.25 |
| S.D. | 25.68 | 22.28 | 28.56 |
| | F 2,55 = 0.41, N.S. | | |
| n moles/mg. Platelet Protein/hr | | | |
| Mean | 46.05 | 39.55 | 59.62 |
| S.D. | 45.95 | 37.30 | 69.95 |
| | F = 2, 53 = 0.51, N.S. | | |

The sample was divided into two groups according to presence or absence of auditory hallucinations. There were 17 patients who presented with auditory hallucinations at the time of inclusion in the study or later during hospitalization. 9 patients did not have any auditory hallucinations at any time. The two groups did not differ significantly, nor were any differences observed from controls.

Two groups were formed – Chronic Paranoid (CP) schizophrenia (N = 11) and chronic undifferentiated schizophrenia (CU) (N = 15) according to RDC (Spitzer et al 1978). There are no significant differences in the platelet MAO activity between the two groups and controls and among themselves.

The sample was then looked at by categorising it into paranoid and non-paranoid

**Table 6**
**Comparison between subgroups of schizophrenia (According to RDC, Spitzer et al 1978) on MAO values**

| | Chronic paranoid (N = 11) | Chronic Undifferentiated (N = 15) | Control (N = 30) |
|---|---|---|---|
| n Moles/$10^8$ Platelets/hr | | | |
| Mean | 32.61 | 17.95 | 30.25 |
| S.D. | 32.40 | 15.12 | 28.56 |
| | F 2.55 = 1.26, N.S. | | |
| n moles/mg platelet protein/hr | | | |
| Mean | 54.49 | 35.96 | 59.62 |
| S.D. | 46.95 | 39.50 | 69.95 |
| | F 2.53 = 0.48, N.S. | | |

**Table 7**
**Comparison of MAO values according to subgroups of schizophrenia (Potkin et al 1978)**

| | Paranoid (N = 19) | Non-paranoid (N = 7) | Controls (N = 30) |
|---|---|---|---|
| n Moles/$10^8$ platelets/hr | | | |
| Mean | 28.74 | 12.83 | 30.25 |
| S.D. | 27.28 | 11.21 | 28.56 |
| | F 2.55 = 1.18, N.S. | | |
| n Moles/mg platelet protein/hr | | | |
| Mean | 55.46 | 12.14 | 59.62 |
| S.D. | 45.19 | 6.68 | 69.95 |
| | F 2.53 = 1.87, N.S. | | |

groups according to criteria described by Potkin and associates (1978). According to these criteria paranoid group includes patients of CP Schizophrenia (RDC) and CU schizophrenia (RDC) with secondary paranoid features of suspiciousness, preoccupation with and systematization of delusions, delusions of control, stereotyped behaviour, intact memory, rigidity of ideas and communication characterised by evasiveness and misleading responses. There were 19 patients in paranoid group and 7 patients in non-paranoid group. Patients in non-paranoid group tended to have lower

values as compared to paranoid group as well as controls but this difference was not statistically significant.

## DISCUSSION

We observed no significant difference in platelet MAO activity between patient and control groups. These observations are in confirmity with observations of 16 previous studies (Shaskan and Becker, 1975, Brockington *et al* 1976, Schildkraut *et al* 1976, White *et al* 1976, Belmaker *et al* 1976, Belmaker *et al* 1977, Sullivan *et al* 1977, Demisch *et al* 1977, Groshong *et al* 1978, Friedhoff *et al* 1978, Landowski, 1977, Banki, 1978, Mann and Thomas, 1979, Meltzer *et al* 1980, Ho *et al* 1982, Karson *et al* 1983). Of the 26 studies observing significant reduction of platelet MAO activity in chronic schizophrenics as compared to controls 6 have been conducted at the same laboratory (Murphy and Wyatt, 1972, Wyatt *et al* 1973, Murphy *et al*, 1974, Berget *et al* 1978, Wyatt *et al* 1978, Potkin *et al* 1978) and have been published more than once. Discrepancy in literature on the subject has arisen probably because of lack of control of several variables namely sex, heterogenous diagnostic criteria, neuroleptic treatment, presence of anaemia and use of different substrates. Most of these have been done on chronic institutionalised patients being treated with neuroleptic drugs over a prolonged period. Well defined diagnostic criteria have been used in only few of them. Studies with women have not been controlled for their menstrual cycle. Presence of anaemia has not been looked into in most of them, except two (Barrettini *et al* 1978, Brockington *et al* 1976). Brockington and associates (1976) excluded anaemic subjects and did not observe any significant difference in platelet MAO activity between patients and controls. Murphy and co-workers (1982) have stated that neuroleptic treatement does not seem to influence platelet MAO activity but their observations were based on repeat estimations after a small drug free period of only 2 weeks on chronically treated patients. Prospective studies on the subject have strongly suggested a contribution of neuroleptic drugs in reducing platelet MAO activity of schizophrenic patients treated for a long time (Becker and Shaskan, 1977, Owen *et al* 1981, Meltzer *et al* 1980, Friedhoff *et al* 1978, Ho *et al* 1982).

The only Indian study on the subject published till date (Sengupta *et al* 1981) was also conducted on a small group of chronic schizophrenics (N = 14) most of whom were being treated with neuroleptic drugs, and females were not controlled for period of menstural cycle. They have also used the spectrophotometric method of enzyme estimation because radioactive labelled substrates are not available in India at present.

We have controlled all of these variables by selecting patients by well defined criteria (RDC, Spitzer *et al* 1978), taking up only males, including fairly age matched controls, excluding presence of anaemia, and excluding drug abuse. 21 out of our 26 patients had not received any neuroleptic treatment. 5 treated patients tended to have lower platelet MAO activity as compared to untreated group as well as controls, though this difference did not achieve statistical significance. The wide range of values observed in our sample is in harmony with observations reported in some other studies (Brockington*et al* 1976, Friedhoff *et al* 1978, Mann and Thomas, 1979; Ho *et al*, 1982; Belmaker *et al* 1977) though variations are much wider in our sample. It could be so because our sample was not controlled for diet. Blood glucose levels have been observed to be correlated with platelet MAO activity (Demisch *et al* 1979). Ideal would have been to keep the patients fasting, but it was difficult in our set-up to keep the psychiatric patients fasting for an

adequate period of time. Another possibility is the effect of wide variations in environmental temperature. Though samples were always carried and processed in ice, there are still chances of artefacts creeping in because of this. Parellel running of samples from patients and controls should have countered this artefact.

Studies of brain monoamine oxidase activity done on chronic schizophrenic patients post-mortem (Reveley *et al* 1981, Utena *et al* 1968, Vogal *et al* 1969, Domino *et al* 1973, Nies *et al* 1974, Schwartz *et al* 1974, Crowe *et al* 1978) have not reported any significant difference between chronic schizophrenics and controls. These studies also support our observations.

Yet another possiblility responsible for non-conformity of literature on the subject is a presence of biological heterogeneity within the schizophrenia group and may be there is a small yet unidentified sub-group of patients that have low platelet MAO activity. Still more investigations are required with better control for several variables as delineated above to identfy this subgroup if it exists. Jeste and associates (1982) have suggested that probably paranoid group of patients form such a sub-group but our observations do not support their observations.

Studies with monozygotic and dizygotic twins (Nies *et al* 1973) and families of chronic schizophrenics (Berrettini *et al* 1980) have suggested that probably platelet MAO activity is genetically controlled and may serve as a genetic marker for vulnerability to chronic schizophrenia. We attempted to study this aspect using single sibling paradigm as suggested by Reider and Gershon (1978). Though we observe a significant correlation between platelet MAO activity of patients and their male first degree relatives, the importance of this observation is nullified by the absence of any significantly lowered platelet MAO activity in the patient group which is a prerequisite before the single sibling paradigm of Reider and Gershon (1978) can be applied.

## Conclusions

There is no significant difference in platelet MAO activity between schizophrenics and healthy controls.

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