Journal of Phylogenetics & Evolutionary Biology

Cocchi et al., J Phylogen Evolution Biol 2014, 2:2
DOI: 10.4172/2329-9002.1000128

Research Article Open Access

Human and Animal Brain Phospholipids Fatty Acids, Evolution and Mood Disorders

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Abstract

The purpose of this paper is to connect the experimental evidence concerning brain phospholipids fatty acids composition by comparing the first warm-blooded animal in the phylogeny (birds) with the human brain at various ages of life (from the fetal period until the eightieth year of age).

The particularity of our investigation is an almost unique opportunity for groped a hypothesis about the evolutionary aspects of the behavior of brain and consciousness, as represented in the human and animal world, as a result of the evidence that led to the diagnostic classification of mood disorders in humans, in their similarity with some animal species. A logical sequence of considerations about the mood disorder diagnosis, due to unequivocal evidence by the use of mathematical tools that cannot be manipulated, it leads to results that most probably indicate and suggest the existence of a common brain "biochemical house", in man and animal. This "common house" will become more and more complex, during evolution, from animal to man, respecting the concept of the molecular equilibrium and allowing to each living being the adaptation to their needs and their roles. Small deviations from the biochemical equilibrium of brain fatty acids can manifest pathological behavioral responses, much amplified. Everything seems to be witnessed by the strong classificatory correspondence of the platelets fatty acids which correspond to psycho pathologies, especially for the Linoleic acid and alpha Linolenic acid, in particular the Linoleic Acid, which, to varying percentages, it may correspond to psychopathological phenomena.

Keywords: Brain; Phospholipids; Fatty acids; Human and animal; Evolution; Consciousness

Introduction

Background of the research

Aware that a molecular involvement of the cell membrane could be expression of a psychiatric disorder, we have tried to understand and explain this phenomenon.

The intention was to study the platelet fatty acids composition in normal and depressed subjects [1-7] and animals [8], because of their similarity to neurons [9-19].

Membrane platelet fatty acids of subjects with clinical diagnosis of Major Depression versus apparently normal subjects have been assessed. The complexity of the membrane dynamics has also suggested the study by means of non-linear advanced analytical tools. In particular, it seemed more appropriate to use Artificial Neural Networks: the Self organizing Map (SOM) - Kohonen Network [20-22].

This particular algorithm allows viewing the result graphically, building a two-dimensional map which places the subjects in a continuous way, not dichotomized. The values of fatty acids of the 2 populations have been administered to the SOM, mixing normal and pathological subjects and hiding the information on to their own pathological condition. As a result, the SOM, called by us ADAM, was able to map the two populations using 3 specific fatty acids, Palmitic Acid (PA-C16:0), Linoleic Acid (LA-C18:2 n-6) and Arachidonic Acid (AA-C20:4 n-6), which represented the majority of total membrane fatty acids, recognizing as similar those belonging to the same population and then separating the normal from the pathological [1]. Several different SOMs have been tested and all of them gave, essentially, the same result. However, the SOM used gave superior information by allowing the results to be described in a two-dimensional plane with potentially informative border areas. The central property of the SOM is that it forms a nonlinear projection of a high-dimensional data manifold on a regular, low-dimensional grid.

The Experimental Evidences

The first experiment was performed by recruiting the subjects, healthy or pathologic, without taking account of specific criteria. No attempt was made of hyper-selecting the sample, according to the recruitment rules of the Evidence Based Medicine (EBM) in the knowledge that, in psychiatry, we must act in a different way [23,24], especially if the goal is the search for strong biomarkers.

The first experiment allowed us to recognize the subject with Mood Disorder from supposed healthy subjects (Figure 1).

Recently we have run a second experiment which allowed us to diagnose Bipolar Disorder and Major Depression [25], (Figures 2-4).

Figure 2 shows the pathologic subjects Major Depression and Bipolar Disorder) all together. Figure 3 (a new SOM has been realized) shows, clearly, that it was possible to distinguish the subjects with Major Depression (red) from those one with Bipolar Disorder (blue). Figure 4 shows the same picture of Figure 3 pointing out an intermediate area which collects both cases (Major Depression and Bipolar Disorder). For each subject we have calculated an Index called B2, We have obtained the B2 Index by the sum of the percentages of each fatty acids (AA, LA and PA), multiplied for the melting point and divided for the molecular weight. B2 is negative for subjects with Major Depression, positive for subjects with Bipolar Disorder. In this way it is possible to recognize also the cases that are within a very close range as showed in Figure 5.

As can be seen, the cases, also within a very close range, are clearly

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Received June 11, 2014; **Accepted** July 14, 2014; **Published** July 21, 2014

Citation: Cocchi M, Tonello L, Gabrielli F, Minuto C (2014) Human and Animal Brain Phospholipids Fatty Acids, Evolution and Mood Disorders. J Phylogen Evolution Biol 2: 128. doi:10.4172/2329-9002.1000128

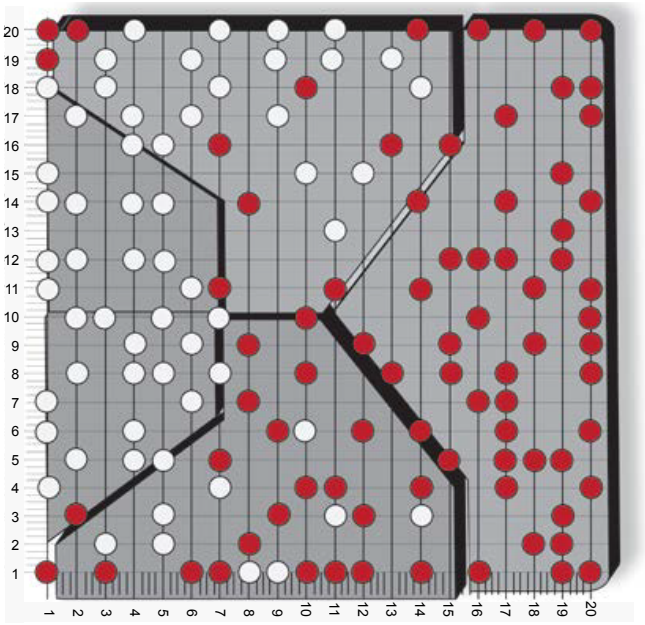


Figure 1: Distribution of all cases [apparently Normal (white) and Pathologic (red)] over the SOM.

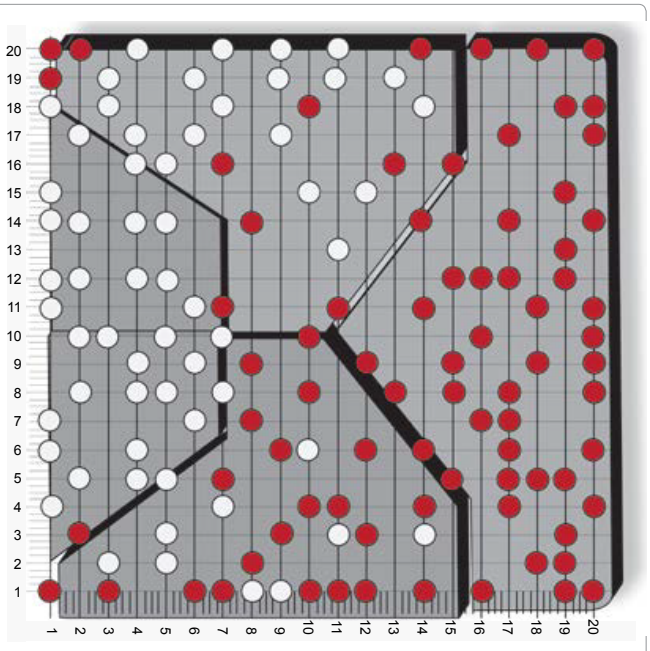


Figure 2: shows the pathologic subjects Major Depression and Bipolar Disorder) all together.

distinguishable. The combination of the SOM and of the B2 index is able to perform the right diagnosis [25]. During the last experiment [25] the psychiatrists have provided us with eight cases of "Suicidal Ideation". When we have classified them over the SOM, the Figure 6 was obtained.

The cases were collected where the SOM recognizes the minimum of Linoleic Acid. In particular seven cases were Bipolar and one with Major Depression, confirming that both can have suicidal ideation and can attempt suicide [25]. The subject, in position 15:4, was uncertain at the psychiatric evaluation; in effect his position is a little bit out from the critical area of the minimum of Linoleic Acid. In the same way, other areas has been found within the SOM (Figure 7): OCD area, Major Depression area, Bipolar area (the largest), Psychotic area etc.

All the experimental findings in humans and animals are resumed in Figures 7 and 8.

According to the psychiatric diagnosis (when definitive) we can recognize: 1= OCD area, 2= Major Depression area, 3= Bipolar area (the largest), 4= Suicide area, 5= Psychotic area, N= apparently normal area. N area collects about the 50% of the sample of subjects considered apparently normal.

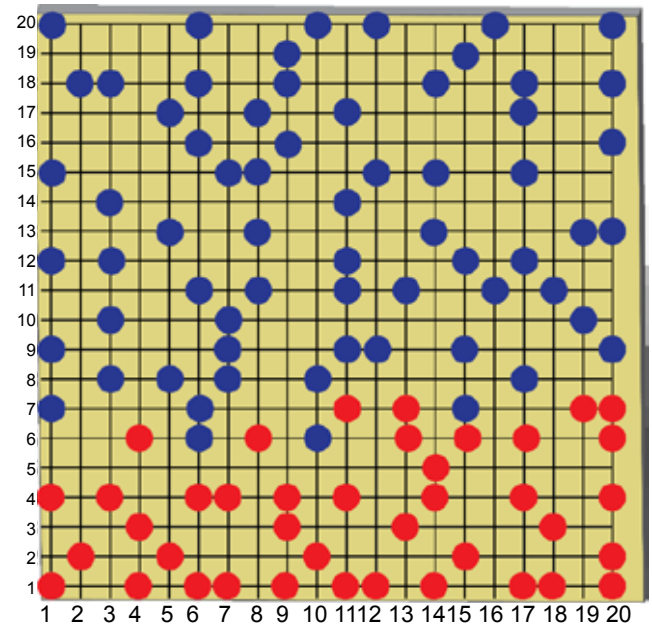


Figure 3: (a new SOM has been realized) shows, clearly, that it was possible to distinguish the subjects with Major Depression (red) from those one with Bipolar Disorder (blue).

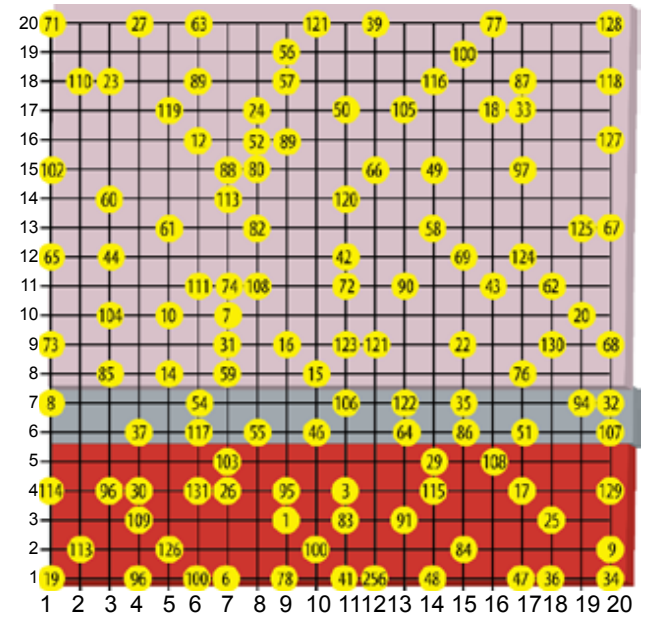


Figure 4: shows the same picture of Figure 3 pointing out an intermediate area which collects both cases (Major Depression and Bipolar Disorder).

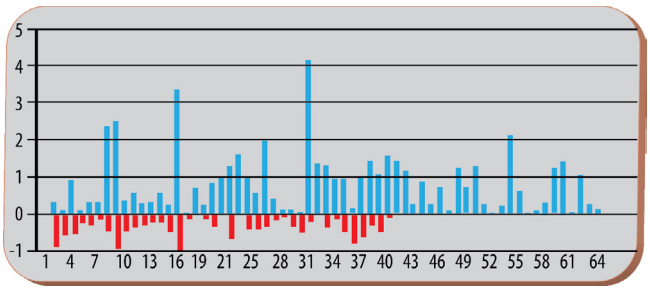


Figure 5: In Figure 5 is represented the classification of the subjects with Major Depression (B2 negative-red) and Bipolar Disorder (B2 positive-blue).

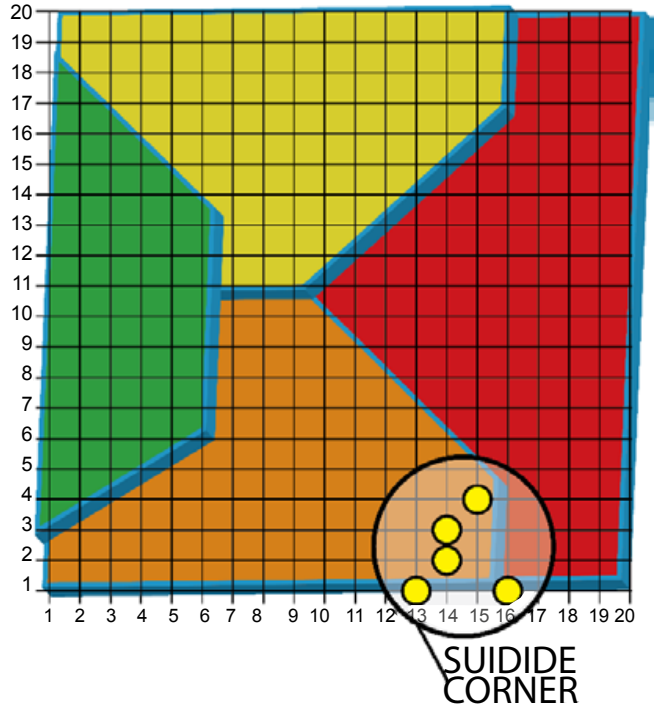


Figure 6: Distribution of the "suicidal" cases over the SOM.

Several different animals have been mapped on the SOM, as well. The molecular similarities [26], observed between animal and man. Figure 8, concern the conditions of Major Depression, Bipolar Disorder, Obsessive Compulsive Disorder.

To confirm the molecular correspondence between man and animal, observe how, e.g. Cat, Bovine, Horse and Donkey, correspond to the area of maximum Linoleic Acid and of Obsessive Compulsive Disorder.

This area is recognized as the point of maximum concentration of Linoleic not only for diagnostics correspondence, but also because it contains the cat, who, as feline, is known to possess desaturase, but with low activity [27], therefore not to be able to transform Linoleic Acid into Arachidonic Acid, resulting in savings of Linoleic, and long living animals [28]. Further, in the same animals, symptoms of OCD can occur [29,30].

Towards Phylogenetic and Evolutionary Considerations

We made a diagnosis of 5 different psychiatric conditions using platelets fatty acids (Figure 5). Because each psychiatric disorder diagnosed represents a different state of consciousness, we hypothesize that this could confirm a basic, common molecular pathway for both, psychiatric disorder and consciousness.

We reached this conviction by retesting all the experiments performed and the molecular evidences that supported the possibility of a relationship between platelet and brain, as well as a comparison between the characteristics of the membrane fatty acids of animal and human brain, being known the central role of the cell membrane in intra-and extra-cellular function and in conditioning the ion channels.

Brain phospholipid fractions and their fatty acids

In the course of research carried out at the Scottish Agricultural College (Cocchi and Noble, data not published), we faced the problem of verifying the fatty acid composition of the phospholipid fractions of the chick embryo brain during development until the first day of life, getting the following result Table 1 and Supplementary Figures 1a- 4a. This result

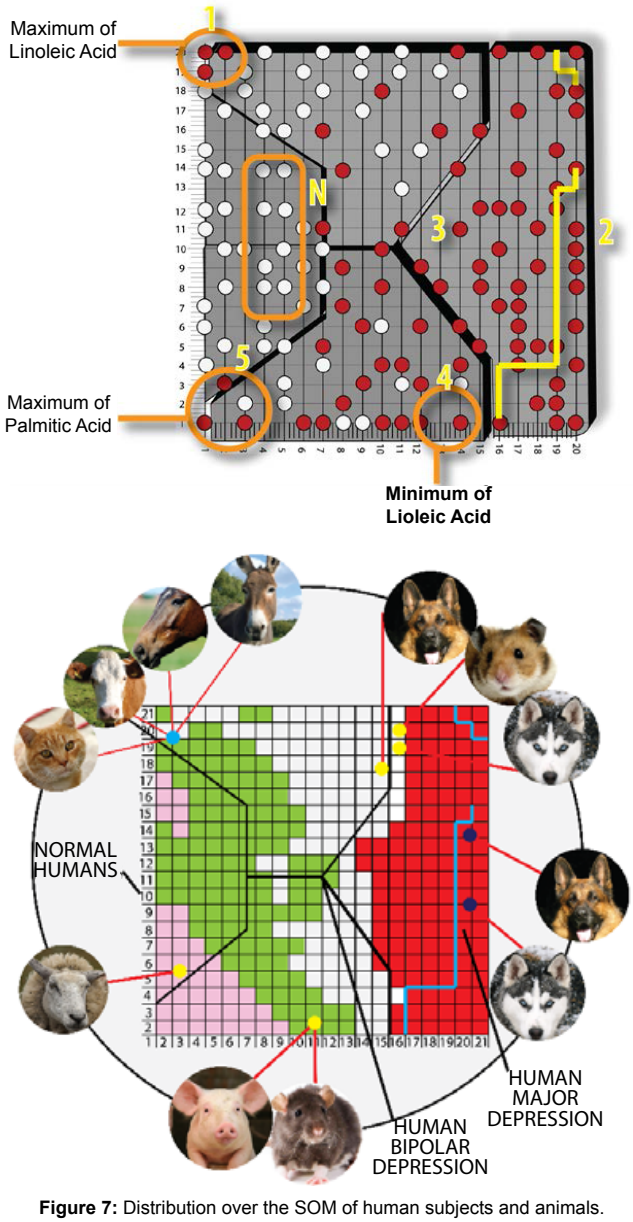


Figure 7: Distribution over the SOM of human subjects and animals.

Phosphatidyl - Choline					
	D12	D14	D16	D19	D22
C16:0	59.06 2.08	61.38 1.08	59.30 0.99	57.19 2.40	56.15 1.62
C16:1	2.19 1.27	ND -	2.00 1.15	ND -	ND -
C18:0	4.81 0.11	3.77 0.84	4.82 0.25	7.91 0.77	10.66 0.91
C18:1	22.67 0.22	23.83 0.23	22.94 0.54	24.66 1.54	25.78 0.14
C18:2	2.58 0.11	2.63 0.07	2.75 0.10	2.21 0.13	2.58 0.01
C18:3	0.25 0.02	0.18 0.03	0.20 0.01	0.27 0.09	0.62 0.07
C20:4	2.89 0.21	3.02 0.10	2.58 0.26	3.25 0.33	2.13 0.21
C22:6	3.48 0.38	4.09 0.21	4.21 0.18	4.00 0.17	1.65 0.47

Phosphatidyl - Serine					
	D12	D14	D16	D19	D22
C16:0	8.91 0.49	10.06 0.49	13.85 1.48	24.22 5.52	9.20 0.22
C16:1	0.30 0.30	0.70 0.70	0.19 0.19	10.38 0.90	ND -
C18:0	35.83 1.00	37.41 0.96	37.33 0.62	25.89 4.39	47.86 1.56
C18:1	8.69 0.79	7.00 0.30	7.88 0.56	13.65 1.69	17.45 0.03
C18:2	1.85 0.34	1.38 0.28	1.51 0.28	2.02 0.45	2.28 0.03
C18:3	0.29 0.03	0.17 0.01	0.17 0.06	0.43 0.01	1.15 0.02
C20:4	3.08 0.20	3.16 0.20	4.14 0.35	2.11 0.81	2.73 0.03
C22:6	39.39 0.92	39.69 0.62	33.89 1.01	18.13 4.61	18.72 1.68

Phosphatidyl - Ethanolamine					
	D12	D14	D16	D19	D22
C16:0	14.54 0.67	13.46 1.24	15.42 1.63	12.60 0.81	9.96 0.16
C18:0	20.04 1.03	21.36 0.51	22.27 0.52	25.33 1.49	31.14 1.65
C18:1	10.20 0.57	8.99 0.35	9.18 0.14	11.67 2.08	18.92 0.55
C18:2	0.89 0.02	0.87 0.04	1.20 0.04	0.66 0.22	1.51 0.07
C18:3	0.31 0.01	0.23 0.01	0.26 0.01	0.28 0.10	1.28 0.13
C20:4	12.72 0.12	12.97 0.42	12.46 0.39	13.74 0.82	14.24 0.78
C22:6	38.96 1.35	40.46 0.93	37.45 1.54	35.36 2.01	22.68 0.39

Phosphatidyl - Inositol					
	D12	D14	D16	D19	D22
C16:0	6.22 0.66	3.53 0.53	10.15 1.82	9.39 1.83	7.82 0.06
C18:0	36.32 0.31	37.61 0.89	35.28 6.67	37.92 1.41	38.36 1.26
C18:1	11.97 0.58	9.02 0.94	19.75 3.26	11.73 0.86	19.03 0.40
C18:2	0.78 0.06	0.62 0.17	1.74 0.68	0.97 0.22	5.64 0.35
C18:3	ND -	ND -	0.10 0.10	ND ND	0.92 0.25
C20:4	40.46 0.27	45.00 0.63	26.05 8.97	35.15 2.40	23.28 0.60
C22:6	3.10 0.60	3.83 0.56	6.44 0.87	4.04 1.14	3.00 0.73

Table 1: Cocchi and Noble, data not published (% of fatty acids of phospholipid fractions during chick embryo brain development). D=Days of chick embryo development Average of 4 sample for each analysis and S.E.

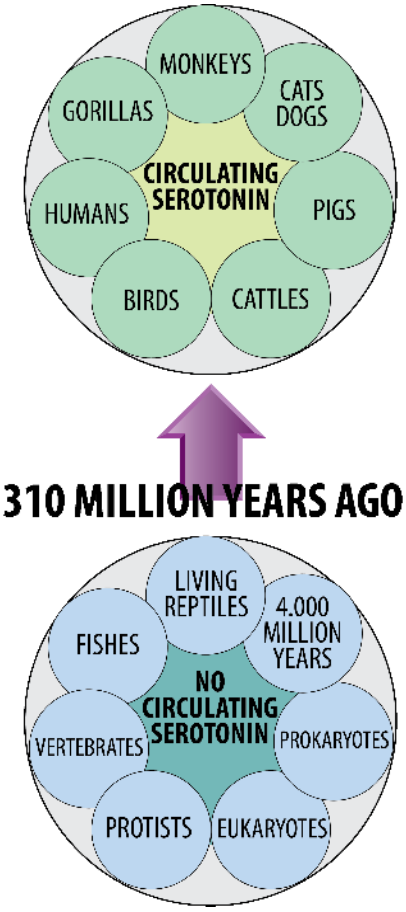
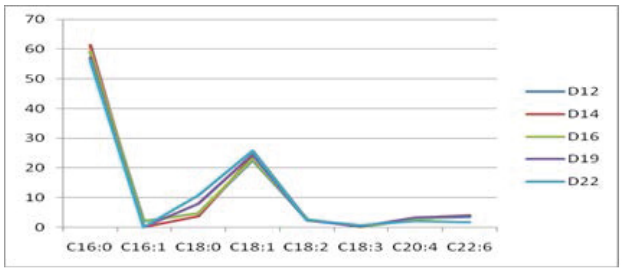


Figure 8: Point of transition between the animals without circulating serotonin and animals with circulating serotonin respectively, cold blooded animals and warm blooded animals.

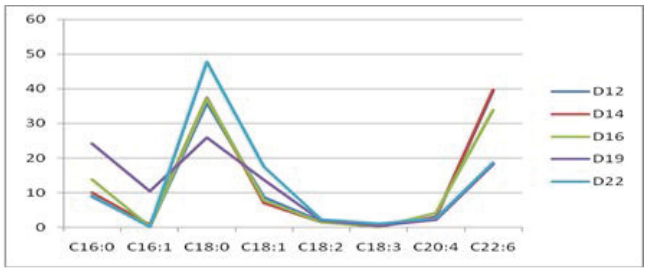
has been compared, now, with the work performed by Svennerholm [31] in human brains of persons not deceased for brain related reasons Supplementary Figures 1b-4b; 1c- 4c; 1d- 4d. Supplementary Figures 1e-8e shows the comparison of the fatty acids of the phospholipid fractions during chick embryo and human fetus development.

As a further demonstration of the constancy of the composition of fatty acids in the brain, in particular as regards the linoleic acid and alpha Linolenic acid, Table 2 and Figure 9 show the fatty acid profile of the pig brain as animal very distant from the chicken and very close, metabolically, to the man [1].

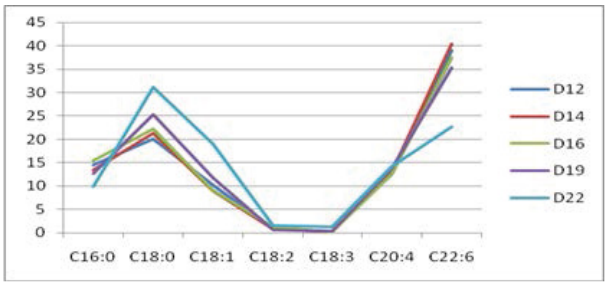
Phosphatidyl - Choline (Figure 1a)



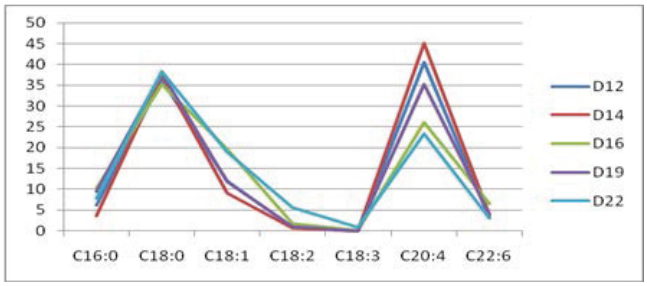
Phosphatidyl - Serine (Figure 2a)



Phosphatidyl - Ethanolamine (Figure 3a)

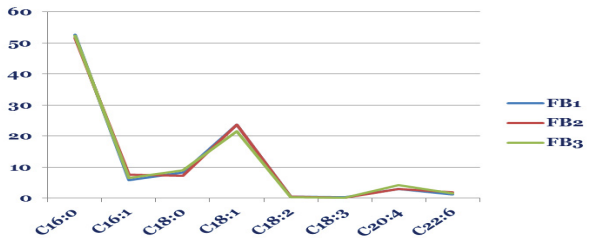


Phosphatidyl – Inositol (Figure 4a)

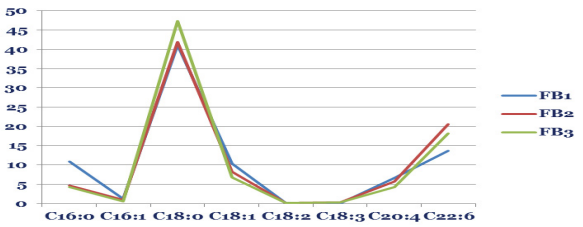


Figures show the fatty acids of the brain phospholipid fractions during chick embryo development until the first day of hatching

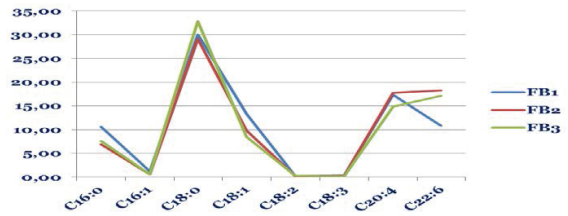
Phosphatidyl – Choline Human Gray Matter (Figure 1b)



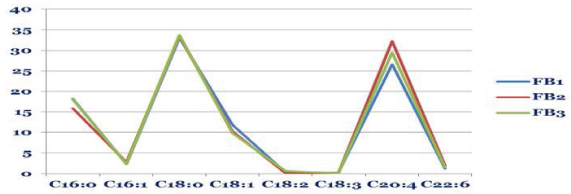
Phosphatidyl - Serine Human Gray Matter (Figure 2b)



Phosphatidyl - Ethanolamine Human Gray Matter (Figure 3b)

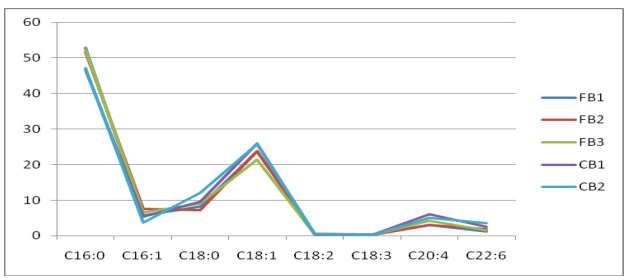


Phosphatidyl – Inositol Human Gray Matter (Figure 4b)

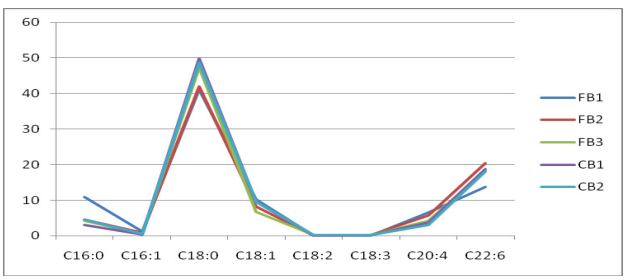


Figures show the fatty acids of the phospholipid fractions (Human Gray Matter) during human fetus development (12 wk old fetus [FB 1], 35 wk old premature fetus [FB 2], 38 wk old premature fetus [FB 3].

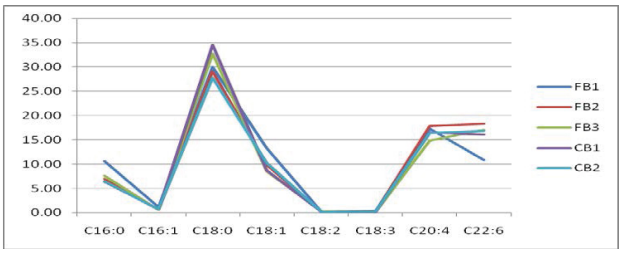
Phosphatidyl – Choline Human Gray Matter (Figure 1c) from fetal age to 1 month (CB1) and 7 months (CB2)



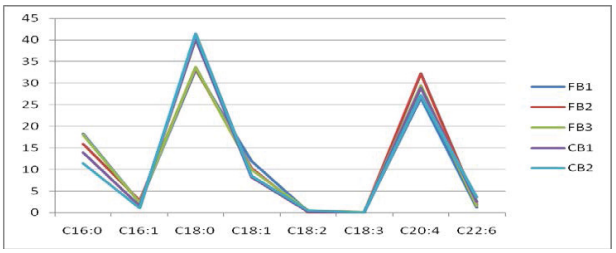
Phosphatidyl – Serine Human Gray Matter (Figure 2c) from fetal age to 1 month (CB1) and 7 months (CB2)



Phosphatidyl – Ethanolamine Human Gray Matter (Figure 3c) from fetal age to 1month (CB1) and 7 months (CB2)

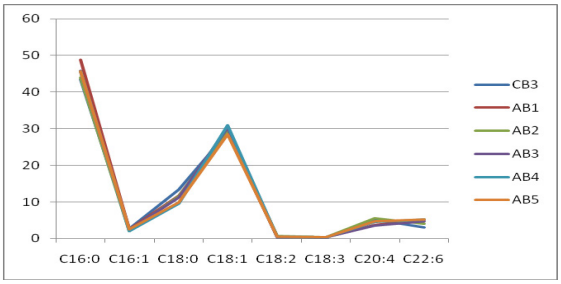


Phosphatidyl – Inositol Human Gray Matter (Figure 4c) from fetal age to 1 month (CB1) and 7 months (CB2)

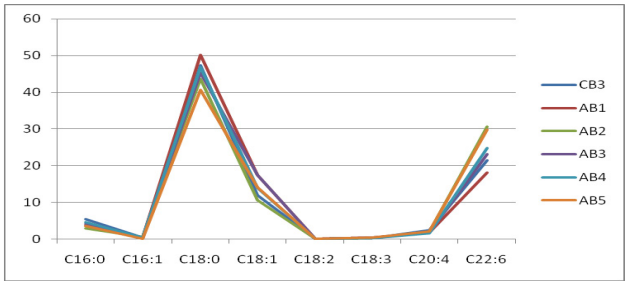


Figures show the fatty acids of the phospholipid fractions (Human Gray Matter) during human fetus development (12 wk old fetus [FB 1], 35 wk old premature fetus [FB 2], 38 wk old premature fetus [FB 3] and at 1 month (CB1) and 7 months (CB2)

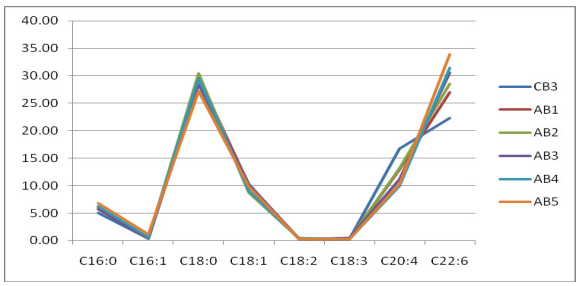
Phosphatidyl – Coline Human Gray Matter (Figure 1 d)



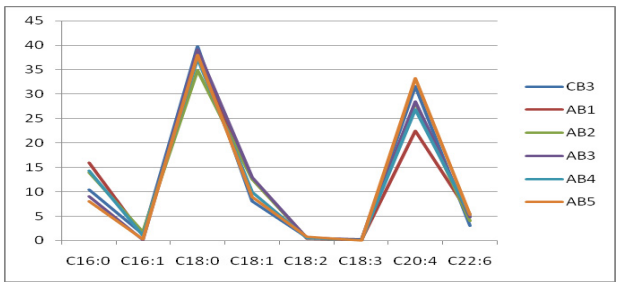
Phosphatidyl – Serine Human Gray Matter (Figure 2 d)



Phosphatidyl – Ethanolamine Human Gray Matter (Figure 3 d)

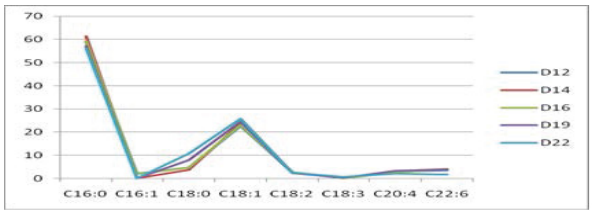


Phosphatidyl – Inositol Human Gray Matter (Figure 4 d)

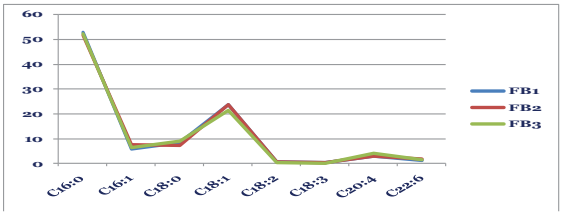


Figures show the fatty acids of the phospholipid fractions (Human Gray Matter) from the age of 4 to the age of 82 [4 yr old female (CB 3), 16 yr old male (AB 1), 26 yr old female (AB 2), 52 yr old female (AB 3), 81 yr old male (AB 4), 82 y-r old male (AB 5)]

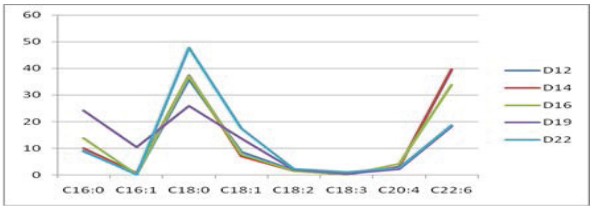
Phosphatidyl – Choline fatty acids, chick embryo (Figure 1e)



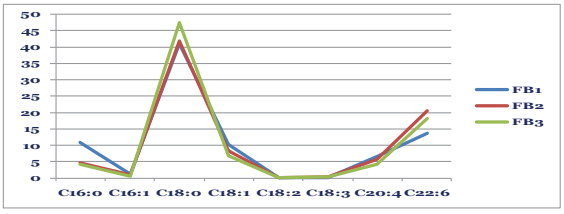
Phosphatidyl – Choline fatty acids, Human Gray Matter (Figure 2 e)



Phosphatidyl – Serine fatty acids, chick embryo (Figure 3e)

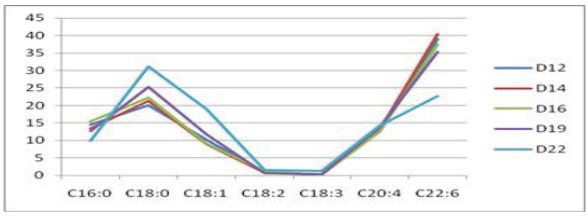


Phosphatidyl – Serine fatty acids, Human Gray Matter (Figure 4e)

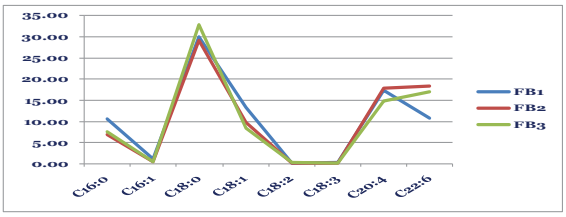


Phosphatidyl – Ethanolamine fatty acids, chick embryo (Figure 5 e)

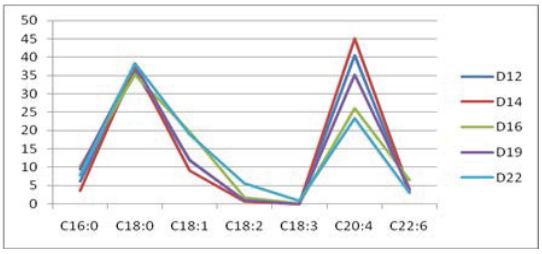
Phosphatidyl – Ethanolamine fatty acids, Human Gray Matter (Figure 6e)

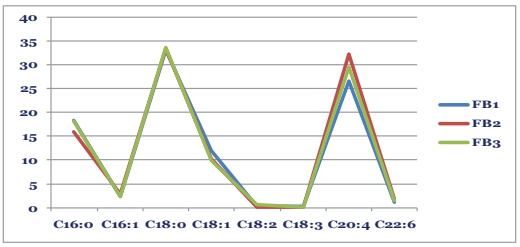


Phosphatidyl – Inositol fatty acids, chick embryo (Figure 7e)



Phosphatidyl – Inositol fatty acids, Human Gray Matter (Figure 8e)





Figures 1, 2, 3, 4, 5, 6, 7, 8 e show the comparison between the fatty acids of the phospholipid fractions during the chick embryo development and the human fetus development.

C16:0	C16:1n9	C18:0	C18:1n9	C18:2n6	C18:3n3	C20:4n6	C22:6n3
12.43	0.47	17.07	18.16	0.31	0.28	7.32	5.93
0.39	0.04	0.37	0.51	0.05	0.04	0.25	0.29

Table 2: Brain fatty acids values of pig (% and SD).

Figure 9: Figure 9 e. Brain fatty acid profile of pig

Table 2 shows the average of the total brain fatty acids (40 samples) of the pig and figure 9 e the linear graph of the main fatty acids. Despite for the pig there are only the values of the total brain fatty acids and not of the phospholipids fractions, it is evident that the linoleic acid and alpha linolenic acid are invariable both in the individual phospholipid fractions and as expression of the total lipid fatty acids.

16:0	18:0	16:1	18:1	18:2 n-6	20:3 n-6	20:4 n-6	22:4 n-6	18:3 n-3	20:5 n-3	22:5 n-3	22:6 n-3
11.9	22.23	0.19	11.16	24.97	0.46	14.36	0.37	0.27	0.27	1.75	9.45
1.18	2.05	0.30	0.97	3.03	0.08	1.07	0.06	0.06	0.06	0.26	1.38

Table 3: Heart phospholipid fatty acids composition (rat).

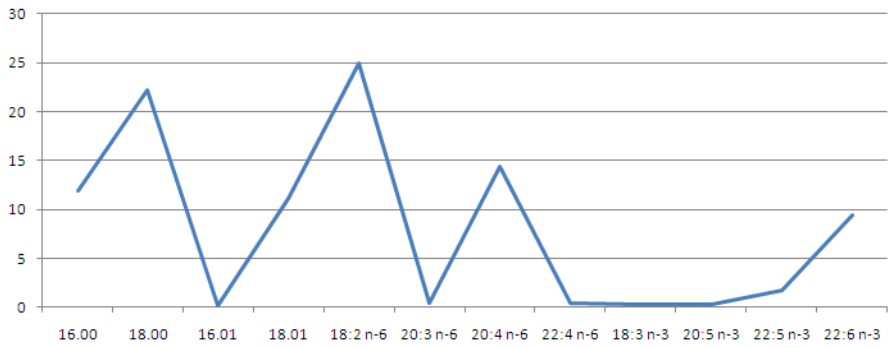


Figure 10: Figure shows clearly the difference between the membrane phospholipids fatty acid compositions of the miocardial cell and of the brain.

C14:0	C16:0	C16:1	C17:1	C18:0	C18:1	C18:1	C18:2	C18:3	C20:3	C20:4	C22:4	C22:5	C22:6
0.87	20.68	1.48	0.81	11.23	22.19	1.82	19.41	0.48	2.11	14.06	1.62	1.16	2.09
0.59	2.15	0.71	0.54	3.00	2.08	0.64	2.69	0.17	0.76	2.41	0.70	0.62	0.80

Table 4: Platelets phospholipid fatty acids composition (Humans).

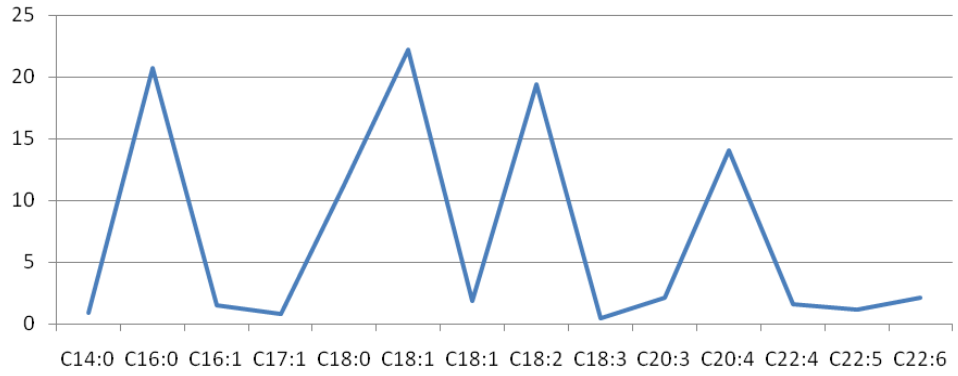


Figure 11: Figure shows, clearly, the difference between the membrane phospholipids fatty acid compositions of the platelet and of the brain.

As an example that highlights the difference in the fatty acid composition of membrane phospholipids, between the brain and other cells is shown in Tables 3, 4, Figures 9 and 10 [32,33].

The comparison between the fatty acids of the brain phospholipid fractions (chick embryo development and human cerebral cortex), at different stages of life, with particular reference to the embryo fetal age of both (man and chicken) show a striking similarity. Of particular interest, is the figure of linoleic acid and alpha linolenic acid, which are present in very low concentrations, despite being essential.

They seem to be, even for their intermediate position in the fatty acids chain, the elements that give structural stability to the membrane, especially in the brain, confirming, with their very low concentration, close to zero, the difficulty to swing towards different concentrations but, also, that subtle modifications of the concentrations can result in huge changes of the molecular elements of neuronal cooperation (interactome).

This particular finding seems to presume, also, the existence of a very selective mechanism of incorporation of the two fatty acids.

A further observation can be made with regard to the circumstance that all the brains (humans and animals) have the same lipid cell membrane structure, considering that the chicken (bird), is the first warm-blooded living being in the phylogeny and the man, the last Figure 9 [33].

Additionally, having identified in different animal species an extraordinary correspondence with the mood disorders that occur in humans, and having full knowledge that each disorder will manifest with different states of consciousness [34,35], it would seem plausible to say that brain and consciousness are sharing a common, basic, molecular aspect. With increasing evolutionary complexity, of course, different and more complex responses occur, corresponding to different expressions of the language, each for his own species.

In particular we want underline the SOM position of OCD subjects (area of maximum concentration of Linoleic Acid), within the normal subjects, according to Marazziti et al. [36] for the characteristics described for these kind of patients: "*The aggressive features observed in OCD patients were more similar to healthy controls than to depressed patients. This suggests that aggression in OCD is a complex phenomenon that probably requires specific instruments of evaluation*".

Discussion

Considerations on the psychopathology in humans versus animals

One possible interpretation of the reduced concentration of Linoleic Acid and alpha Linolenic acid in brain phospholipids, and their compositional similarity in animal species to man, can be interpreted on the need to maintain a stability of the membranes of nervous elements and, therefore, of the same, whole, brain[Cocchi and Noble, data not published [31].

The mechanisms of production of polyunsaturated fatty acids are sensitive to temperature [37,38] for the phenomena that it induces: to its decrease, increases the production of polyunsaturated fatty acids, to its increase, the opposite phenomenon occurs.

If these phenomena are related to the amount of serotonin at the synaptic level, it is well known that in depression, reduces the availability, low amount of serotonin, a characteristic of the phenomenon of depression, could correspond to a decrease in temperature, and then confirms, consistently, the finding of increased production of arachidonic acid [39].

All this, is explicable with the experimental results which showed that, the increase of the membrane fluidity, reduces of the uptake of serotonin (for decreased exposure of the receptors) and, in the case of an increase in the viscosity of the membrane (for a greater exposure of receptors) increases the serotonin uptake [40,41].

The observation of the similarity of the brain fatty acid composition of the phospholipid fractions, in animals and humans, together with the finding of a very small amount of linoleic acid and alpha linolenic acid, would be, therefore, to guarantee that the temperature oscillations, even if contained in physiological ranges do not correspond to the processing of amount of the substrate, such as to induce profound changes in the levels of viscosity and fluidity with the known consequences.

The lipid structure of the brain as well as investigated (Cocchi and Noble, data not published, [31]) manifests the same characteristics in the extreme positions of the evolution of warm-blooded animal (from birds to humans) in the course of phylogeny.

This evidence allows us to consider that the brains of all animals, including humans, have a "common biochemical pattern" and, at the same time, this could also explain the correspondence of the molecular aspects of psychopathology, between man and animal.

It is possible to assume, reasonably, that while the manifestation of Mood Disorders is expressed by the increase or decrease of specific fatty acids, linoleic acid and alpha linolenic acid, especially linoleic acid, is confirmed, even in its consolidated stability, as the element capable of inducing, for small changes, amplifications of pathological brain responses.

The face of evidence of molecular contiguity between man and animal with regard to mood disorders and also in the knowledge that they are expressed in different language modes, it remains to be further explored the actual interpretation of this phenomenon.

The psychopathological aspects investigated for the man and, correspondingly, to the animal, are, in the ordinary sense, a disease. In animals, it is plausible that the same events correspond to a characteristic of the species.

What could be the border between pathology and characteristic of the species? A border impalpable or absence of institutional classification? Probably, even for the man you could talk about a characteristic of species that determines the need for specific roles within the society?

The aspects of the molecular contiguity of humans and animals psychopathological expression recognize themselves in a common biochemical picture. What should make us think that the psychopathology of man is a real disease and that, on the contrary, in the animal is a characteristic of the species? A difficult question to answer. Would the level of complexity reached by man makes him able to use reasoned tools of defense against the society and of the society towards him?

The complexity of the human brain and the expression of these diseases have required the need to organize the clinical approach to the pathology. The psychopathology of the animal, if considered characteristic of the species, differently would engage, its common molecular similarity, in the management of itself within the habitat, in order to ensure the needs that are proper to him and the most useful strategies for survival.

The major difference between the psychopathological problem in man and animal may consist mainly in the fact that, over time, man has been organized to recognize and treat the expression of psychopathology in an attempt to alleviate the suffering of the individual, and, for some psychopathological expressions, to intervene with respect to the social context.

The psychopathological condition of the animal, however, might correspond to specific roles for each species, in achieving its strategic programs in the environmental context, which could be a condition required for the life. In recent times it has begun to develop a discipline that deals with the psychological problems of the animal, which attempts to cure them, recognizing a state of discomfort to the animal and the same human rights.

It would address, in this way, the problem of a characteristic of the species that could enhance the psychopathological expression of the animal when it is in contact with the epigenetic phenomenon that is represented by its relationship with man, risking of losing its characteristic of species.

If we consider the distribution of the various species of animals in the SOM built for the assessment of psychopathological states of man, we realize that, at least for the animals studied, and also considering the dog as the animal most similar to man by the behavioral point of view, we don't find a representative of any of them in the area which corresponds to severe psychosis or suicidal ideation.

We find, instead, a representation of the animal world in the area of major depression, bipolar disorder and obsessive compulsive disorder (aggression), like if the evolutionary complexity had saved structuring characteristics of these animals' species enabling them to survive expressing the most appropriate behavior.

What can be evaluated as true psychopathology of the human brain, in the general condition of depression or bipolar disorder, might be the suicidal ideation and the severe psychosis.

These two pathological extremes are heavily influenced, biochemically, by particular characteristics of the concentration of linoleic acid, which expresses the highest concentration in the obsessive compulsive disorder and the minimum concentration, in the suicidal ideation [25].

These biochemical conditions could represent the basal conditioning elements of the complexity in the molecular cooperation of the neuron.

The difference between animal and man could be, beyond the evolutionary complexation, due, mainly, to the different social environment and/or habitat and to the different language modes, while remaining unchanged the basic principle of the molecular identity.

We like to remember, about animals (canine in particular) the exchange of opinions between Cocchi and Mender [26]:

"One thought that I have is to seek in wolves an olfactory (e. g. urinary pheromonal), body-kinetic, or prosodic route for the expression of mood rather than a linguistic mode. The first mode might be especially fruitful, since canines seem to live in an olfactory sensory world of which we cannot even conceive, much as these creatures most likely cannot conceive of our own human linguistic universe. Can a dog write a polyphonic symphony of subtle odors? Perhaps. Might that composition feel emotionally like a Chopin etude? Why not?"

Conclusion

The essential acknowledged points, which can be nonetheless subject to further and deeper discussions are:

- The molecular contiguity between human beings and animals, that is the expression of common biochemical features, allowing for an interpretation of reality based on precise biological and language contexts specific to each species, rather than hierarchies;
- The possibility to find links – backed by empirical evidence – between biomolecular quantitative approaches and quantum ones;
- Last but not least, the possibility – thanks to really slight biomolecular variations – to distinguish in a practical way, i.e., from the chemical, physical and mathematical point of view, different psychiatric conditions and, subsequently, different states of consciousness. On the basis of the results obtained, having shown that many animal species are similar, in behavior, to man, we believe it is possible to derive, from the neuron of each animal species, information that may clarify the common and basic molecular mechanisms to understand what is that makes the difference of the increasing degrees of evolutionary complexity.

The evidence of the biochemical data obtained in the classification of the various human psychopathologies, which correspond to the animal ones, let us a final question that could open a new scenario about the relationship between man and animal. It has been proved that different animal species present elements of classification corresponding to those of a single species, i.e. the human being, with the exception of the suicide attempt and severe psychosis. We can ask ourselves if the human species sums up all animal species, at least those studied. Further, suicidal ideation and psychosis are therefore conditions that, as it would be easy to assume, they belong only to the human being?

In other words, man is the only being to have a clear thought of illness and death just because his level of complexity is so high as to synthesize, with qualitative emergencies, all animal species, without falling into the anthropocentric bias?

"The scientist does not study nature because it is useful to do so. He studies because it derives pleasure; and derives pleasure because she is beautiful" [42].

"If nature was not beautiful, it would not be worth knowing, and life would not be worth living "[43].

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