What role can psychological and psychiatric research play for our understanding of the brain’s networks? Conversely, what can the research on neural networks offer psychiatry?

massimiliano.aragona@uniroma1.it
• “Negative” part: a philosophical critique
  – History of Psychiatry
  – Epistemology of Psychiatry
  – Phenomenology/Hermeneutics

• “Positive” part: a few examples
The crisis of Psychiatry

Only a couple of decades have passed, but those already seem “good old days”. Much of that enthusiasm and faith has now vanished [...] the questions I am now receiving from journalists [...] focus not so much on “new developments in the manual” (the most common question when the DSM-IV was launched) as on [...] “why we produce this classification at all, since we do not have a solid ground on which to base it”

Maj (2012, p.161), commenting his involvement in the creation of the DSM-5.
“... the neo-Kraepelinian paradigm established by Robins and Guze and institutionalized in the DSM has resulted in so many problems and inconsistencies that a crisis of confidence has become widespread. [This drives] a transition from a period of normal science [...] to a period of extraordinary science”

Zachar & Jablensky, 2014, p.9-10
Being in crisis, Psychiatry (in its present form) cannot contribute to Brain Networks

But the question is: why psychiatry is in crisis? What is wrong with it?

If we understand the problems, we can make the necessary corrections in order to give a real contribution to Neuroscience

Being in a period of “extraordinary science” we are free to explore creative solutions
• The DSM-III as the beginning of a paradigmatic phase in psychiatric nosography
• The DSM as a system in crisis
• Waiting for a revolution
Anomaly: a seemingly empirical problem (something arising in the research data that impede scientific progress) which is the consequence of the way the system is structured
Some DSM anomalies

Internal Heterogeneity

100 cases of Disorder X

50 cases of Disorder X

Drug to be tested

Randomization

50 cases of Disorder X

Placebo
Some DSM anomalies

Internal Heterogeneity

Similarly, when we select a group of patients with Disorder X and we search for common underlying neurobiological or neurocognitive dysfunctions.

If the group is highly heterogeneous, it is difficult to find out a common basis of the disorder.
Some DSM anomalies

Comorbidity

If one patient has disorder A + B + C + D and another patient has disorder A + E + F, can we recruit them for a study of disorder A?

If we do, the group will be heterogeneous (the comorbid disorders being uncontrolled intervening variables)

If we select only “pure” cases (those with only disorder A), 90% of ordinary patients will be excluded (low ecological validity)
Some DSM anomalies

Internal Heterogeneity
• Lack of phenomenal determination
• Lack of qualitative hierarchies
• Quantitative thresholds
• Polythetic Criteria

Comorbidity
• Splitting
• Decision to severe phenomena in different chapters
• Lack of phenomenal determination
• Progressive deletion of exclusion criteria
In the more than 30 years since the introduction of the Feighner criteria by Robins and Guze, which eventually led to DSM-III, the goal of validating these syndromes and discovering common etiologies has remained elusive. Despite many proposed candidates, not one laboratory marker has been found to be specific in identifying any of the DSM-defined syndromes. Epidemiologic and clinical studies have shown extremely high rates of comorbidity among the disorders, undermining the hypothesis that the syndromes represent distinct etiologies. Furthermore, epidemiologic studies have shown a high degree of short-term diagnostic instability for many disorders. With regard to treatment, lack of treatment specificity is the rule rather than the exception.
The implicit theoretical stance of the “atheoretical” DSMs

The DSM-III as a “neo-Kraepelinian” classification system
And so, what is the basic similarity between Kraepelin and the DSM-III (and followers)?

“Probably the fundamental point in common among Kraepelin, the neokraepelinians and the DSM-III is the following implicit idea: namely, that by improving the diagnostic procedures real ens of nature could be enucleated, and that from this basis (the individuation of these pathologies at the descriptive level) it would be possible to grasp the underlying level of the causes responsible for them” (Aragona, 2010, p.386)
The key point of the DSM’s “neo-Kraepelinian” crisis
“Of the two major theories underlying the “atheoretical” DSM, namely the neopositivist-derived view on how to operationalize scientific concepts and the neo-Kraepelinian view on objects and purpose of a psychiatric classification, the former is responsible of the emergence of anomalies, whereas the latter seems to be the one that enters in a state of crisis because these anomalies conflict with its basic tenets”

Aragona, 2009, p.32
The DSM-III as the beginning of a paradigmatic phase in psychiatric nosography

Internal heterogeneity and comorbidity as anomalies

Anomalies depend on the way core concepts are defined

Crisis and revolution
Revolutionary Proposals

Diagnosis of Spectra
Dimensional diagnosis
Aetiopathogenetic diagnosis
State of the art

Revolutionary proposals
reductionist “etiopathogenetic” model

As expected, the DSM-5 has not reshaped its axes etiopathogenically; the model remains a neo-Kraepelinian one, based on the phenomenal description of mental disorders fulfilling descriptive and neopositivist operative diagnostic criteria.
State of the art

Where we are

“a bridge to new diagnostic approaches without disrupting current clinical practice or research”
State of the art

Revolutionary proposals
neurocognitive model

Research Domain Criteria (RDoC)
New Classification Framework

"Our expectation... is that identifying syndromes based on pathophysiology will eventually be able to improve outcomes."

State of the art

Toward the future: revolutionary proposals

Bruce A. Future diagnostic systems cannot reflect ongoing advances in genetics, neuroscience, and cognitive neuroscience models. The goal of mental diagnosis: the seven pill Open Access Medicine

7 November 2015

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<thead>
<tr>
<th>Domains/constructs</th>
<th>Units of analysis</th>
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<td><strong>Negative valence systems</strong></td>
<td>Genes</td>
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<td>Potential threat (“anxiety”)</td>
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<td>Sustained threat</td>
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<td>Imitation, theory of mind</td>
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<td>Social dominance</td>
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<td>Facial expression identification</td>
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<td>Attachment/separation fear</td>
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<td><strong>Arousal/regulatory systems</strong></td>
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<td>Arousal and regulation (multiple)</td>
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<td>Resting state activity</td>
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Table I. Research Domain Criteria Matrix. “Circuits” can refer to measurements of particular circuits as studied by neuroimaging techniques, and/or
“Kraepelinian” approach
Mental illness as descriptively enucleated putative ens of nature

Confirmation through the discovery of the underlying neurobiological etiology

RDoC Project
Study of the resulting symptoms when they are dysfunctional

Individuation of the basic neurocognitive functions to be studied
RDoC is necessary, but very oversold

Allen Frances
Department of Psychiatry, Duke University, Durham, NC, USA

The past half century has witnessed heroic advances in the basic sciences of brain research, genetics, and molecular biology. But there has also been a surprising and disappointing paradox: none of the exciting scientific findings has had any impact whatever on the everyday practice of clinical psychiatry. Fortunately, we have available effective treatments for most mental disorders, but there have been no real breakthroughs in our understanding of psychopathology and ways of treating it.

Why the gaping disconnect between a basic science enterprise that is remarkably dynamic and a clinical practice that is relatively static? In fact, psy-

Scientific American

Cross-Check
Critical views of science in the news

Psychiatry in Crisis! Mental Health Director Rejects Psychiatric “Bible” and Replaces with... Nothing
By John Horgan | May 4, 2013 | 67
The proposal of a cognitive neuropsychiatric taxonomy tries to reconcile the personal level of Folk Psychology, the computational level of Cognitive Psychology, and the neuronal level.

Mental disorders might be breakdowns of neurocomputational mechanisms.

At present such an approach cannot provide a systematic reformulation of psychiatric taxonomy, but it could suggest interesting directions for future psychiatric research.
In general, can neurocognitive sciences solve the problems raised by psychiatry?

Shall we expect from neuroscience the advancement of knowledge promised by the “neo-Kraepelinian” biological psychiatry?

What kind of practical impact may we expect?
Provided that after psychotherapeutic interventions neuroscience suggests that some neural correlates are changed, …

“… findings from neuroimaging may produce knowledge to guide psychotherapeutic interventions by specifying what should be stimulated in these individuals in order to normalize deficient neural activities”

Peres & Nasello, 2008
There is little doubt that the recent impressive development of neuroimaging and neurophysiological technologies has resulted in a better knowledge of brain structure and function. However, this has raised new epistemological challenges.

… are mental symptoms reducible to neurocognitive function ‘without residual’? And if a relationship between brain addresses and mental symptoms exists, is it a one-to-one correspondence so that we can hope to discover a specific neurocognitive alteration for every different mental symptom?

Aragona & Marková, *in press*
The Cambridge School

*The hermeneutic level*
Formation and meaning of mental symptoms: history and epistemology
Lecture presented at the Roman Circle of Psychopathology, Rome, Italy, 16th February 2012

GERMAN ELIAS BERRIOS
Chair of Epistemology of Psychiatry, Robinson College, University of Cambridge (UK)

Historical evidence shows that mental symptoms were constructed in a particular historical and cultural context (19th Century alienism). According to the Cambridge model of symptom-formation, mental symptoms are mental acts whereby sufferers configure, by means of cultural templates, information invading their awareness. This information, which can be of biological or semantic origin, is pre-conceptual and pre-linguistic and to be understood and communicated requires formatting and linguistic collocation. Mental symptoms are hybrid objects, that is, blends of inchoate biological or symbolic signals and cultural configurators. ‘Culture’ plays a very deep role in symptom-formation because templates can attenuate or abolish the specificity of the biological signals involved. This means that signals from different brain sites can be configured as the same symptom and signals from the same site as different symptoms. Although always present, the neurobiological substratum is not fundamental in the understanding and management of mental symptoms. These can only be comprehended in relation to the manner of their construction and the cognitive and emotional biographies of each patient. Direct interference with the brain sites involved may dull mental symptoms but is unlikely to offer long-term cure. If the configuratory style and needs of the patient are not understood and dealt with, he is likely to keep re-constituting or replicating his symptoms in relation to other biological signals. In summary, mental symptoms are not passive happenings but genuine mental acts. Hence, the manner and motivation of their construction may be more important than the signal of brain distress that might have provoked them in the first place.

Keywords: philosophy of psychiatry, epistemology of psychiatry, psychopathology, history of psychiatry

DIAL PHIL MENT NEURO SCI 2013; 6(2): 39-48
7 Neuroimaging in psychiatry: Epistemological considerations 112
Ivana S. Marková and German E. Berrios

Alternative Perspectives on Psychiatric Validation

Edited by Peter Zachar, Drozdstoj St. Stoyanov, Massimiliano Aragano, and Assen Jablenski

- Tackles a vitally important topic in mental health – one seldom given the attention it deserves
- Includes chapters by some of the leading philosophers, psychiatrists, and psychologists of our day
- Places these issues within an historical context

978-0-19-968073-3
November 2014 | Paperback
STATISTICAL CORRELATION BETWEEN PROXY VARIABLES REPRESENTING THE BRAIN AND PROXY VARIABLES REPRESENTING MENTAL PHENOMENA

SCORES DERIVED FROM RATING SCALES

IN PSYCHIATRY, ‘SCIENCE-MAKING’ IS BASED ON CORRELATIONS

Berrios, modified
The resolution power of proxy variables representing the brain is increasing at a rapid pace. Proxy variables representing mental states & behaviours have remained static since their construction during the 19thC. To gain practical meaning correlations require at least a partial match of resolution power. The proxyhood of Psychopathology needs refining. This work should start with mental symptoms.
CAMBRIDGE MODEL FOR SYMPTOM-FORMATION

Brain signal → ‘Construction’ → symptom-expression

Personal Construction
e. styles of talking about the body: personal, familial, social, cultural, etc.

Co-construction within a ‘Dialogical Encounter’

Mental Symptom

Berrios, modified
Biological signal

CULTURAL configurators

Envelope 1

Berrios, modified
Biological signal
Self-interpretation
Biological signal

Self-interpretation

Dialogical negotiation

Envelope 1

Envelope 2

Berrios, modified
Biological signal

Self-interpretation

Dialogical negotiation

Size of ‘informational aperture’?
Mental symptoms with primary brain inscriptions are those which correspond in time and space with the brain activity that gives rise to them. Here there would appear to be a direct and specific relationship between the biological and the “semantic.” For example, an ictal focus or brain lesion in a particular area might directly trigger organic hallucinations. [...] The sense of the symptom in these cases would be carried predominantly by the “biological” element and thus have less in the way of a meaningful connection for the individual. In other words, irrespective of how the symptom is expressed, it can be viewed as more stereotypical and relatively empty from personal significance.

Marková & Berrios, 2014
On the other hand, mental symptoms with secondary brain inscriptions are those symptoms where the relationship between the biological and “semantic” is not direct. Here, the brain representations can be viewed as simply the concomitant neurobiology substratum. In other words, the biological is the non-specific brain activity that accompanies mental activity. In this case, the sense of the symptom is carried predominantly by its “semantic” element.

Marková & Berrios, 2014
NUMERICAL PROXYHOOD OF THE OBJECTS OF PSYCHIATRY

Loss of Information

Slippage of information

Complaint as experienced
Observation, Selection, Interpretation by clinician
'Description' in words
Conversion to scale item
Numbers as entered into correlations

Statistical correlation with proxy variables representing the brain

Conceptual distance

Berrios
To sum up:
Independently from technical/methodological limitations of neurocognitive, neurophysiological & neuroimaging techniques, there are basic limitations arising from within psychology & psychiatry. They include:

• Heterogeneity & Comorbidity: unsuitable samples

• Quality of the proxy variables to be correlated

• Size of ‘informational aperture’ and hermeneutic envelopes
Part 2: a few examples

E.g. 1: Direct stimulation of brain circuits
Pathways from cannabis to psychosis: a review of the evidence

Jonathan K. Burns*
Department of Psychiatry, Nelson R Mandela School of Medicine, University of KwaZulu-Natal, Durban, South Africa

The nature of the relationship between cannabis use (CU) and psychosis is complex and remains unclear. Researchers and clinicians remain divided regarding key issues such as whether or not cannabis is an independent cause of psychosis and schizophrenia. This paper reviews the field in detail, examining questions of causality, the neurobiological basis for such causality and for differential inter-individual risk, the clinical and cognitive features of psychosis in cannabis users, and patterns of course and outcome of psychosis in the context of CU. The author proposes two major pathways from cannabis to psychosis based on a differentiation between early-initiated lifelong CU and a scenario where vulnerable individuals without a lifelong pattern of use consume cannabis over a relatively brief period of time just prior to psychosis onset. Additional key factors determining the clinical and neurobiological manifestation of psychosis as well as course and outcome in cannabis users include: underlying genetic and developmental vulnerability to schizophrenia-spectrum disorders; and whether or not CU ceases or continues after the onset of psychosis. Finally, methodological guidelines are presented for future research aimed at both elucidating the pathways that lead from cannabis to psychosis and clarifying the long-term outcome of the disorder in those who have a history of using cannabis.

Keywords: Cannabis, psychosis, schizophrenia, causality, neurobiology, cognition, outcome
Part 2: a few examples

E.g. 1: Direct stimulation of brain circuits

Psychopathological and Cognitive Effects of Therapeutic Cannabinoids in Multiple Sclerosis: A Double-Blind, Placebo Controlled, Crossover Study

Massimiliano Aragona, MD,* Emanuela Onesti, MD,† Valentina Tomassini, MD,‡
Antonella Conte, MD,‡ Shiva Gupta, MD,§ Francesca Gilio, MD,‡ Patrizia Pantano, MD,‡
Carlo Pozzilli, PhD,‡ and Maurizio Inghilleri, PhD, MD‡

Objectives: To study possible psychopathological symptoms and cognitive deficits, abuse induction, as well as general tolerability and effects on quality of life, fatigue and motor function in cannabis-naïve patients with multiple sclerosis (MS) treated with a free-dose cannabis plant extract (Sativex). Experimental studies reporting transient positive and negative psychotic symptoms together with other psychopathological phenomena, is greater in subjects predisposed for psychosis and probably in adolescent smokers due to a greater vulnerability of the developing brain. On the other hand, cannabis use has been frequently reported in anxiety disorders: it might
Psychopathological and Cognitive Effects of Therapeutic Cannabinoids in Multiple Sclerosis: A Double-Blind, Placebo Controlled, Crossover Study

Massimiliano Aragona, MD,* Emanuela Onesti, MD,† Valentina Tomassini, MD,‡
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Objectives: To study possible psychopathological symptoms and cognitive deficits, abuse induction, as well as general tolerability and effects on quality of life, fatigue and motor function in cannabis-naïve patients with multiple sclerosis (MS) treated with a free-dose cannabis plant extract (Sativex).

In conclusion, cannabinoids prescribed for therapeutic reasons to cannabis-naïve patients with MS did not induce onset of psychotic or anxiety symptoms and did not impair cognition. Safety and tolerability were generally good, drug tolerance and dose increasing were not reported during the trial, and desire for Sativex or abuse was not present at follow-up. However, the positive correlation between blood levels of Δ-9-THC and psychopathological scores suggests that at dosages higher than those used in therapeutic settings, interpersonal sensitivity, aggressiveness, and paranoid features might arise. Finally, the effects of cannabinoids on quality of

Clinical Neuropharmacology • Volume 32, Number 1, January/February 2009
Part 2: a few examples
E.g. 1: Direct stimulation of brain circuits
Part 2: a few examples

E.g. 1: Direct stimulation of brain circuits
Part 2: a few examples

E.g. 1: Direct stimulation of brain circuits
Part 2: a few examples
E.g.2: Eating Disorders
Part 2: a few examples
E.g.2: Eating Disorders

Psychotic phenomena in Binge Eating Disorder: an exploratory MMPI-2 study

Massimiliano Aragona, Anna Maria Petta, Andrea Balbi

Conclusions. At least in some patients, there might be an overlap between some psychotic basic phenomena (disordered sense of basic Self, of bodily experiences, and hyperreflectivity), and those basic disturbances in identity development and Self-schemas which are at the base of eating disorders.

Archives of Psychiatry and Psychotherapy, 2015; 2: 13–20
DOI: 10.12740/APP/43321
Part 2: a few examples
E.g.2: Eating Disorders

Different levels, different instruments
- General Psychopathology – clinical assessment, SCL-90
- Eating behaviors – clinical and nutritional assessment, Three Factor Eating Questionnaire, Binge Scale Questionnaire, Eat-26
- Body Image - Body Uneasiness Test
- Self-Identity - Identity and Eating Disorders Questionnaire (IDEA)
- Basic Self-Disturbances – Items from the Bonn Scale of Basic Symptoms and Parnas et al.’s Anomalous Self Experiences
- Cognitive basic features – Neuropsychological and Neurocognitive ‘ecological’ tests
- Psychophysiological correlates - heart rate variability
Part 2: a few examples
E.g.3: Empathy
The many faces of empathy, between phenomenology and neuroscience

Massimiliano Aragona, Georgios D. Kotzalidis, Antonella Puzella

Summary

The definition of empathy differs among the domains which deal with it. Introduced in medicine and psychology in the late 19th-early 20th century, it received contrasting definitions from philosophers and psychopathologists. The neuroscience paradigm of empathy for pain allowed us to identify two components of empathy, one automatic, bottom-up, and one cognitive, top-down. The role of mirror neurons in this context appears to be central. Empathy is influenced by perception of other, closeness, belonging to a social group, and gender, with women empathizing more than men. The areas involved are the self-other distinction areas (dorsomedial prefrontal cortex and temporoparietal junction), the anterior insula, and the anterior cingulate. The activations identified in the brain allow for better understanding the phenomenon, but not to draw a consensus definition. Rather than providing responses, the neurosciences send back to philosophy new, formidable questions to be asked.

Archives of Psychiatry and Psychotherapy, 2013; 4 : 5–12
The mirror neurons
Mirror neurons: the basis of the imitations of emotions

- Significantly increased activity in the right amygdale either when the subject observes and imitates the facial expression of emotions (fMRI study)

  - Carr et al. (2003) *PNAS* 100:5497-5502
Empathy and the “pain matrix”

Empathy for Pain Involves the Affective but not Sensory Components of Pain

Tania Singer,1* Ben Seymour,1 John O’Doherty,1 Holger Kaube,2 Raymond J. Dolan,1 Chris D. Frith1

Our ability to have an experience of another’s pain is characteristic of empathy. Using functional imaging, we assessed brain activity while volunteers experienced a painful stimulus and compared it to that elicited when they observed a signal indicating that their loved one—present in the same room—was receiving a similar pain stimulus. Bilateral anterior insula (AI), rostral anterior cingulate cortex (ACC), brainstem, and cerebellum were activated when subjects received pain and also by a signal that a loved one experienced pain. AI and ACC activation correlated with individual empathy scores. Activity in the posterior insula/secondary somatosensory cortex, the sensorimotor cortex (SI/MI), and the caudal ACC was specific to receiving pain. Thus, a neural response in AI and rostral ACC, activated in common for “self” and “other” conditions, suggests that the neural substrate for empathic experience does not involve the entire “pain matrix.” We conclude that only that part of the pain network associated with its affective qualities, but not its sensory qualities, mediates empathy.
The process of embodied simulation is:

- Implicit, automatic, and unconscious
- It enables the observer to use his/her own resources to penetrate the world of the other without explicitly theorizing about it

Bottom-up process

The Roots of Empathy: The Shared Manifold Hypothesis and the Neural Basis of Intersubjectivity

Vittorio Gallese

Dipartimento di Neuroscienze, Sezione di Fisiologia, University of Parma, Parma, Italy
The complexity of empathy within neuroscience

The Neural Substrate of Human Empathy: Effects of Perspective-taking and Cognitive Appraisal

Claus Lamm\textsuperscript{1,3}, C. Daniel Batson\textsuperscript{2}, and Jean Decety\textsuperscript{1,3}

feelings. Depending on how empathy is triggered, an automatic tendency to mimic the expressions and motivational processes, which influence whether observing a conspecific in need of help will result in empathic concern, an important instigator for helping behavior.
The complexity of empathy within neuroscience

“Every instantiation of mirroring is always a process in which others’ behavior is metabolized by and filtered through the observer’s idiosyncratic past experiences, capacities, and mental attitudes. More studies will have to investigate the relationship between personality traits and the quantitative and qualitative nature of mirroring mechanisms [...] This occurs in a nonconscious, predeclarative fashion, though modulated by our own personal history, that is, by the quality of our attachment relations and by our sociocultural background”

Empathy, Embodied Simulation, and the Brain: Commentary on Aragno and Zepf/Hartmann
Vittorio Gallese
J Am Psychoanal Assoc 2008; 56; 769
The complexity of empathy within neuroscience

“Every instantiation of mirroring is always a process in which others’ behavior is metabolized by and filtered through the observer’s idiosyncratic past experiences, capacities, and mental attitudes. More studies will have to investigate the relationship between personality traits and the quantitative and qualitative nature of mirroring mechanisms […] This occurs in a nonconscious, predeclarative fashion, though modulated by our own personal history, that is, by the quality of our attachment relations and by our sociocultural background”
Temporal dynamic of neural mechanisms involved in empathy for pain: An event-related brain potential study

Yan Fan\textsuperscript{a,b}, Shihui Han\textsuperscript{a,b,*}

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Received 25 May 2007; received in revised form 28 July 2007; accepted 30 July 2007
Available online 6 August 2007

Abstract

Previous neuroimaging studies have identified a neural circuit that is involved in empathy for pain. However, the temporal dynamics of neural activities underlying empathic processes remains poorly understood. This was investigated in the current study by recording event-related brain potentials (ERPs) from healthy adults who were presented with pictures or cartoons of hands that were in painful or neutral situations. Subjects performed a pain judgment task that required attention to painful cues in the stimuli or a counting task that withdrew their attention from these cues. The ERP results showed early differentiation between painful and neutral stimuli over the frontal lobe at 140 ms after sensory stimulation. A long-latency empathic response was observed after 380 ms over the central–parietal regions and was more salient over the left than right hemispheres. The early and late empathic responses were, respectively, modulated by contextual reality of stimuli and by top-down attention to the pain cues. Moreover, the mean ERP amplitudes at 140–180 ms were correlated with subjective reports of the degree of perceived pain of others and of self-unpleasantness. The ERP results support a model of empathy for pain consisting of early emotional sharing and late cognitive evaluation.

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\textit{Keywords:} Empathy; ERP; Pain; Attention; Contextual reality

1. Introduction

Successful social interactions require the capacity to understand and interpret the emotional world of others. Understanding this world is a complex process that involves the identification of others’ emotional states, the appreciation of their subjective experience, the ability to engage in perspective-taking and the expression of empathy. Empathy is often considered to be an essential component of social understanding and interaction (Decety and Chaminade, 2006). A theory of mind approach helps to comprehend subjective emotional reaction to others’ feeling, the results of these studies could be influenced by intention of self-presentation and social desirability. Early...
The complexity of empathy within neuroscience

“The ERP results showed early differentiation between painful and neutral stimuli over the frontal lobe at 140 ms after sensory stimulation. A long latency empathic response was observed after 380 ms over the central–parietal regions and was more salient over the left than right hemispheres. The early and late empathic responses were, respectively, modulated by contextual reality of stimuli and by top-down attention to the pain cues.”

Fan & Han, 2008
Within Neuroscience, empathy is shifting from a simple automatic bottom-up process to a more complex phenomenon in which different mechanisms (either bottom-up and top-down processing) are involved.
Future research will differentiate the components of the empathic process, in a recursive interaction between phenomenological descriptions of the experienced components of empathy and neuroscientific progressive detection of subcomponents and interactive modulations of the process.
• Psychopathology will be helpful in this process because it may show how empathic phenomena vary in different persons, in different psychopathological conditions, and in different interactional contexts
Thanks!!

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Dialogues in Philosophy, Mental and Neuro Sciences

http://www.crossingdialogues.com/journal.htm