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Explanatory organization and psychiatric resilience: Challenges to a mechanistic approach to mental disorders

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Abstract This contribution aims to address epistemological issues at the crossroads of philosophy of science and psychiatry by reflecting on the notions of organization and resilience. Referring to the debate on the notion of "organization" and its explanatory relevance in philosophical neo-mechanistic theories, I consider how such positions hold up when tentatively applied to the mental health context. More specifically, I show how reflections on psychiatric resilience, cognitive reserve, and accommodation strategies challenge attempts to embrace a mechanistic perspective on mental disorders. A deeper focus on these aspects of mental health is relevant to theoretical discussions on explanatory models as well as for clinical practice, diagnosis, and treatment.
KEYWORDS: Psychiatric resilience; Cognitive Reserve; Organization; Mechanism

Riassunto *Il ruolo esplicativo dell'organizzazione e la resilienza psichiatrica: sfide ad un approccio meccanicistico alla malattia mentale* – Questo articolo intende discutere alcuni temi epistemologici all'incrocio tra la filosofia della scienza e la psichiatria, riflettendo sulle nozioni di organizzazione e resilienza. Muovendo dalle riflessioni sulla nozione di "organizzazione" sviluppate nell'ambito delle teorie neo-meccaniciste, e dalla difesa del suo ruolo esplicativo, il lavoro analizza la possibilità di estendere alcune posizioni relative alla spiegazione scientifica al contesto delle scienze della salute mentale. Più nello specifico, si illustra come le indagini sulla resilienza psichiatrica, la riserva cognitiva e le strategie di compensazione possano mettere in discussione i tentativi di abbracciare una prospettiva meccanicistica quando si affronta il tema del disturbo psichiatrico. Una maggiore attenzione ai suddetti filoni di ricerca può avere un impatto significativo sia sulla discussione relativa ai modelli teorici di spiegazione della malattia mentale sia sui processi clinici di diagnosi e terapia.
PAROLE CHIAVE: Resilienza psichiatrica; Riserva cognitiva; Organizzazione; Meccanicismo

Introduction: Disorders and the notion of organization

THE PRESENT WORK FOCUSES ON the possible role of psychiatric resilience and cognitive reserve in mechanistic explanations for mental disorders, and how they might influence our understanding of organization as an ex-

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planatory principle. I do not take a stance here on precisely what a psychiatric disorder may ultimately be. While this is surely an extremely important issue that relates to the current discussion, it is not the core concern of the reflections that follow, which instead aim to discuss thorny issues related to mechanistic explanations of mental disorders. For the purposes of this contribution, let me just mention several broadly accepted definitions of mental disorders in the literature. According to Jerome Wakefield, «a disorder is a harmful dysfunction, wherein harmful is a value term based on social norms, and dysfunction is a scientific term referring to the failure of a mental mechanism to perform a natural function for which it was designed by evolution».[1] For Dominic Murphy, «mental illnesses are caused by distinctive pathophysiological processes in the brain»; they «are destructive processes taking place in biological systems», and «a particular destructive process is the way the disease occurs in humans».[2] These definitions refer to mental disorders as *dysfunctional* or *destructive*: diseases (and psychiatric diseases in particular) are characterized as "dysfunctional", "destructive" insofar as they contrast with some standard or the "normal" functioning of the systems at stake.

Epistemological reflections on psychiatric disorders, and related concerns about psychiatric nosologies and explanations, have proliferated over the last few years. Major concerns include the variability of classifications – as shown by different versions of DSM – and difficulties in elaborating explanations which encompass the heterogeneous range of factors involved. Some recent views in the literature have appealed to the notion of mechanism, in order to provide scientific support for a more advanced and stable nosology based on causal explanatory accounts of diseases. The search for causes, and an understanding of their mutual interactions within a mechanism, is invoked to reduce the variability of such definitions and to provide improved nosology. The aim is to go beyond purely symptomatic accounts and align psychiatry with the rest of medicine. This trend is not only evident in the philosophical literature,[3] but also finds some advocates within psychiatry itself. Commenting on the so-called Research Domain Criteria Project initiative,[4] for instance, eminent psychiatrists such as Thomas Insel and Bruce Cuthbert have stressed that investigations are to be pursued «from molecular factors to social determinants – to understand normal and abnormal behaviour, based *on a deep understanding of mechanisms*»,[5] and that we should develop «*a more mechanistic understanding* of how such factors as life events and the social environment interact with development to produce a range of observed outcomes».[6]

Psychiatric diseases can be represented as complex systems that can be described at numerous levels,[7] including, for instance, genetic, neurophysiologic, psychological, social, and economic factors. The standard interactions between factors that normally ensure socially acceptable behaviour no longer hold; instead, divergent arrangements between interacting parts underlie changed behaviour, which is regarded as pathological. If diseases follow a course – from aetiology to the onset of symptoms, to healing/chronicity/death – there must be some overall functioning of the system that – following the definitions noted above – is responsible for global non-functioning, some kind of destructive or dysfunctional behaviour. In other terms, when considering pathologies as mechanisms, we are considering them as systems that disrupt the orchestrated functioning of our organism, or of parts of it, and the resulting behavioural outcomes. Some understanding of how this occurs must be part of the explanatory processes for diseases, understood as conditions which threaten to disrupt forms of stability and equilibrium in the standard functioning of the organism. An explanation of a disease should reveal the factors that contribute to *pathology as a system*, insofar as this system is dysfunctional with respect to the organism considered to be in a condition of health. In the following section, we will consider the role

the notion of "organization" plays in mechanistic perspectives when elaborating explanatory accounts of a well-functioning system.

1 The explanatory role of organization

The idea of organization appears to be crucial to various theories that have found broad consensus in the philosophy of science, and the very definitions of mechanism they put forward. Let us consider some: «Mechanisms are entities and activities organized such that they are productive of regular changes from start-up to finish or termination conditions»;[8] «A mechanism is a structure performing a function in virtue of its component parts, component operations and their organization. The orchestrated functioning of the mechanism is responsible for one or more phenomena»;[9] «A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon».[10] In other terms, the behaviour of a mechanistic system as a whole depends on the exact way in which component parts act and mutually interact. Not only does the notion of organization appear in such definitions, it also helps explain a range of qualitative differences in the performance of active roles within a system. Levy and Bechtel, in particular, claim that «organization involves *an internal division of causal labor* whereby different components *perform different causal roles*».[11] A specific contribution to mechanistic functioning is thus related to a *specific mode of organization*. Whenever (a) different components of the system make different contributions to behaviour, and (b) the differential contributions of these components are integrated, exhibiting *specific interdependencies*, we have organization. Given the role it plays in definitions of mechanisms, and the explanatory aims of mechanistic accounts, organization is considered to be crucial to scientific explanation.

Organization implies a specific division of labour among the variables involved, and is important not only with respect to intralevel functioning, which depends on the properties of entities (or parts), their structures, and their modes of interaction, but also with respect to constitutive explanations, which concern interlevel relations. Its explanatory role is directly addressed, in *How organization explains*, by Jaakko Kuorikoski and Petri Ylikoski, who state: «Constitutive mechanistic explanations explain a property of a whole with the properties of its parts and their organization».[12] To capture both the explanatory relevance of the causal properties of parts and the organizational aspect of mechanistic explanations, Kuorikoski and Ylikoski elaborate on the notion of organization, identifying a range of different dimensions according to which it can be characterized. This leads to a typology of organizational dependence meant to better account for organizational interactions. The three dimensions they suggest for evaluating organization are: diversity in the kinds of components; the network structure between the components; and diversity in the kinds of relations. As for diversity in relations, they claim that the properties of the interactions between the parts include duration, rate, inhibition, promotion, and modulation, among others. These properties of the relations between parts are held to be explanatory with respect to system-level behaviour if we can link changes in the specific properties of such relations to some specific change in system-level properties.

As is widely known, in the last few decades, a debate over mechanisms has been prominent in philosophical discussions on scientific explanation and scientific practice. The psychiatric sciences have not been immune to the considerable success of these neo-mechanistic accounts. As already noted in Section 1, some of the appeal of mechanistic notions is their potential for providing more stable descriptions and classifications. Associating psychiatric diseases with underlying mechanisms could, in principle, allow us to implement causal explanations and

thereby offer more reliable and enduring definitions and nosology. However, while the appeal to mechanisms, interacting entities and levels, and their organization provides some valuable epistemological tools, it also opens up a broad range of concerns. If a system's functioning is warranted by its organization, we must first and foremost ask: *what* organization is relevant for understanding disorders? *What* does organization exactly explain, and what does it not explain, if the relevant organization is far from clear? How fine-grained does our account of organization have to be in order for it to be explanatorily significant? And, in the end, what is organization explanatorily relevant *for*?

As mentioned above, the notion of organization can play a double role in relation to: (i) the mutual intralevel interactions among entities involved; (ii) the hierarchical constitution of the system. Kuorikoski and Ylikoski have stated that organizational explanation is facilitated by looking for ways in which the organization itself can be considered to rely on components that are almost independent, while Craver has claimed that «organization is the inter-level relation between a mechanism as a whole and its components. Lower-level components are made up into higher-level components by organizing them spatially, temporally and actively into something greater than the mere sum of parts».[13] It is worth considering whether this conception in which component parts are organized into some appropriate whole, yet remain localizable and epistemically decomposed, is (always) suitable for psychiatric explanations and which aspects of psychiatric disorders are significant when discussing the explanatory role of organization. Organization is taken as implying some specific form of dependence, which is held to be responsible for some global effect of interest. Discovering the organization of intertwined factors would greatly advance our understanding of mental disorders. However, in most cases, the precise nature of mutual relations between the elements involved may remain somewhat opaque. To address explanatory issues, and the kinds of stability that actually matter for both explanatory accounts and clinical concerns, let us now turn to some recent mental health studies on cognitive reserve and psychiatric resilience.

2 Cognitive reserve and psychiatric resilience

In this section, I zoom in on two concepts that, I believe, deserve deeper attention in the philosophy of psychiatry and provide some interesting epistemological challenges: "cognitive reserve" and "psychiatric resilience". While sometimes addressed together, they are often discussed separately and their descriptions do not always overlap. I will consider some recent work on cognitive reserve and psychiatric resilience and suggest some possible theoretical consequences of this work for the concept of organization and its role in scientific explanation.

In addition to the individual variations that frequently characterize patients, psychiatric approaches take personal accommodation and compensation strategies into account. These are capacities that might – or might not – be activated and interfere with the expression of a pathology, to the point of preserving – to different extents and in different ways – cognitive capacities otherwise at risk. Resistance, accommodation and compensation are responses that do not fully reestablish healthy conditions, nor are they activated *whenever* any disrupting factor is present. Furthermore, they may manifest in differing ways. Concepts such as "cognitive reserve" or "neural compensation" have been introduced to «account for the frequent discrepancy between an individual's measured level of brain pathology and her expected cognitive performance».[14] They are discussed as compensatory and neuroprotective factors, and considered particularly relevant for diseases related to various kinds of brain damage.[15] *Cognitive reserve* stands for differences in cognitive processing due to intellectual ac-

tivity over a lifetime and other environmental factors. It is invoked to explain differential susceptibility to functional impairment due to a pathology or, more generally, neurological impairment. Cognitive reserve is understood to work via *neural compensation*, that is, by utilizing alternative brain networks than those usually engaged for cognitive performance in standard healthy conditions. A wide range of life experiences is considered to contribute to more efficient individual cognitive processing, including levels of educational and occupational attainment (years of education, vocabulary, literacy, ...) and engagement in leisure and social activities. This suggests that neural activity in the present moment has been shaped by the quality of individual cognitive exposures and/or activities across the lifespan. Chanraud and colleagues, for instance, describe cases where people who have stopped drinking alcohol still have very clear signs of damage in the frontal lobe yet score normally in memory tests. Neuroimaging evidence suggests that in such cases the brain compensates for its deficits by recruiting a different pathway to perform a given cognitive task.[16] Others have reported evidence in support of the fact that a significant number of elderly people (over 85), whose scans suggest Alzheimer's, do not show any of the symptoms.[17] What we have, in general, are *non-systematic differences* in the relations between brain damage and functional outcomes: cognitive reserve refers to the capacity of the brain to cope with pathology by finding alternative ways to get things done. Although, cross-sectional studies also indicate that volumetric changes can occur due to prolonged periods of intellectual stimulation – typically associated with higher education and/or specialized occupational skills – cognitive reserve and accommodation are unlikely to be associated with discrete, fully localizable features of the human brain.

Cognitive reserve and neural compensation are introduced to *explain* why patients with relatively similar conditions may nevertheless exhibit very different functional profiles; these constructs can be used as a *predictive* index for cognitive and psychosocial behaviour, but also impact *definitions* and *diagnostics*. In the diagnostic setting, we might focus on performance in cognitive tests or, instead, rely on imaging results to explain individual differences that help preserve cognitive function in the presence of brain pathologies. For instance, in studies of dementia, «the brain reserve capacity (BRC) hypothesis argues that this capacity derives from an individual's unique *neural profile* (e.g., cell count, synaptic connections, brain volume, etc.). Complimentarily, the cognitive reserve (CR) hypothesis emphasizes inter-individual differences in the *effective recruitment of neural networks* and cognitive processes to compensate for age-related effects or pathology».[18]

Cognitive reserve is usually a silent capacity that happens to be called upon when needed. Issues addressed in the literature include difficulties in understanding how cognitive reserve is modulated, and how to measure and, possibly, quantify it experimentally.[19] In fact, studies often rely heavily on the use of proxy measures, such as, e.g., premorbid IQ, education, and occupation. Here, confounders related to data collection are clearly a problem. It may be difficult to measure levels of education, occupational roles, and leisure activity rates in a uniform fashion, while additional factors (e.g., lifestyles and eating habits) may also modulate individual predispositions to cognitive disorders. This further hinders our understanding of whether, how, and to what extent certain putative neural processes underpin cognitive reserve and contribute to the remarkable variability in cognitive outcomes seen with brain disorders, leading to some caveats on the use of proxy measures. Investigations often refer to both intelligence research (e.g. on the fronto-parietal network[20]) and socio-economic factors. Studies on cognitive reserve suggest a neurodevelopmental perspective on psychiatric disorders, taking a long-term view that considers both elements in the patient's past and their prognostic prospects. Debate is also flourishing on whether cognitive reserve

tends to directly compensate for underlying pathologies, or, rather, slows down neuropathological processes.

Some analogous considerations apply if we consider the notion of "psychiatric resilience", defined as «a dynamic process of successful adaptation to stressful experiences or adversity». Measurement issues abound here as well: «while resilience is often operationalized as the absence of psychopathology, operationalizing resilience as a continuous outcome may better capture the wide variation in adaptation following adverse events»[21] than thinking in terms of dichotomous outcomes would allow us to do. Resilience is taken to reflect variation in responses to different kinds of stressful life events. Studying resilient outcomes not only implies a focus on the detrimental results of adverse events, but also encourages a deeper and more general analysis of the full range of individual differences that might be responsible for different degrees of – more or less successful – adaptation to stressful events (e.g. being fired, getting divorced, ...). Studies have been carried out to identify protective factors that predict sustained good mental health, with much interest in predicting outcomes[22] and modes of interventions aimed at enhancing resilience by targeting multiple social and lifestyle factors (e.g. co-parent support, good-quality social relationships, physical exercise, ...). Here too, studies on resilience address different dimensions that impact the capacity to resist disruption to the system and remain close to standard behaviour. For instance, Long and colleagues have investigated how resilience may provide protection from alcohol use disorders (AUDs), trying to shed light on both genetic and environmental causes. Horn and colleagues have focused on post-traumatic stress disorder (PTSD), often triggered by acute stressors, such as trauma, tragedy, or significant threat. Their research intends to identify the genetic, epigenetic, neurochemical, psychosocial, and environmental underpinnings of resilience, and is based on the idea that resilience is mediated by adaptive changes encompassing *several* environmental factors, neural circuits, numerous neurotransmitters, and molecular pathways.[23] Studies on neural substrates mediating stress responses also include the use of animal models, aiming to shed light on potential avenues for pharmacological interventions that could activate resilience processes.[24] However, emerging areas of investigation are beginning to develop new strategies, not just treat disorders. While the focus is generally on pharmacological solutions, they also aim to screen the population and identify at-risk youth and adults as soon as possible. Resilience enhancing measures involve addressing individual, family, and community factors.

Although often addressed separately – as in most of the studies cited above – the notions of cognitive reserve and psychiatric resilience belong to a family of related notions, whose definitions, boundaries, and mutual relations are not very precisely defined or distinct. As pointed out by Watson and Joyce,[25] a whole range of notions and multidimensional constructs can be recalled – such as cognitive reserve, brain reserve, neural reserve, neural compensation, and brain maintenance. Recent studies on neural plasticity show how active engagement of cognitive processes can modify synaptic structure and function even after the age when the brain is fully developed, shaping the neural networks, which in turn mediate cognitive functions so as to use them most efficiently. Debate and puzzles remain concerning the mismatch between analogous underlying conditions and diverse cognitive performances. How do cognitive reserve and psychiatric resilience modulate the impacts of neurodegeneration? This question was originally tackled largely in dementia studies, but later extended to research on, e.g., multiple sclerosis, acquired brain damage, schizophrenia and affective disorders. An additional question of considerable epistemological interest is how these factors should influence our modes of explanation, and our understanding of what counts as explanatorily rel-

evant in accounting for mental disorders? «Resilience is a factor that must be evaluated in every patient and that shall help us determine the outcome of psychiatric disorders and will also be a determinant in the occurrence of relapses».[26]

3 Reflections on explanatory practice: What do organization and resilience explain?

A mechanistic perspective on cognitive reserve and psychiatric resilience can, I believe, stimulate various engaging reflections on their explanatory significance, and psychiatric explanatory practices more generally. The first question worth addressing is: *what* kind of organization is actually relevant from an explanatory standpoint? The idea of organization as identifying a particular division of highly specific causal labour among parts in a mechanistic system is unlikely to prove fruitful. The idea of pursuing some "division" of the systems considered to be engaged by a certain behaviour and of identifying specific diverse causal labour performed by the individual interacting parts – along the lines suggested by, for instance, Levy and Bechtel[27] – proves problematic. While a specific sort of assembly may account for a specific behaviour/functioning, clear-cut epistemic decomposition and/or attempts to specify precise localizations and identify discrete steps do not look promising, at least for the time being. Explaining a certain range of disorders while also taking into account reserve/resilience implies clarifying why a mismatch occurs between expected and actual cognitive performance, and why the organization of lower-level components – i.e. the orchestrated functioning of, for instance, strictly genetic and/or neurophysiologic elements – *may or may not per se make a difference to the explanandum*. Given such a mismatch and the relevant inter-individual differences that are detected, the relation between the functioning of the constituents and global cognitive behaviour still remains in question. Even if, in general, organizational explanation is facilitated by considering a specific organization to be composed of semi-independent parts (through some epistemic decomposition), it is knowing the *specific organizational dependencies*, and their different modes of breaking/resisting, that would allow us to better understand how competition between protective and destructive factors produces variations in symptoms and cognitive performances. Localization and/or decomposition do not provide useful epistemic tools to grasp this kind of distributed functionality, nor have effective computational explanatory strategies been devised so far.

Does a focus on cognitive reserve and psychiatric resilience affect the very conception of the explanandum at stake? Are they a sort of *addendum* to the explanation of the disorder, or are they part and parcel of the explanation itself? What we have is some long-term process – including e.g. education or occupational achievements – which, through some as yet not completely specified and understood means, impact neurobiological functioning and thereby significantly affect cognitive performance. Variables that are described at different levels thus enter the picture, with no current notion of cognitive reserve or psychiatric resilience being entirely and exclusively given in terms of specific low-level sets of entities and their specific relations. We are not given, in other terms, organizational details required by a mechanistic explanatory approach. The phenomena briefly reviewed in section 3 suggest that aspects such as education, occupation and IQ are involved insofar as they can affect the overall cognitive behaviour of the system in cases where stress/disturbance perturbs the system, and that they must therefore be relevant when explaining disorders. Within the context of mechanistic scientific explanation, this cannot but lead to the idea that "organization" plays a fundamental role. But it also highlights the limits of decomposition or localizing strategies. In other terms, this kind of example, while not per se incompatible with any mechanistic account whatsoever, at least challenges some common ways of con-

ceiving mechanisms and mechanistic explanations as mental simulations of how a mechanism's components behave to bring about the target phenomenon. I am not claiming that complete, maximally detailed causal knowledge should be sought in order to have an explanatory account, but rather arguing that *how* the underlying organization is related to the overall cognitive patterns deserves further clarification. While more details are not necessarily better, some deeper analysis is needed, to avoid "organization" from becoming a rather empty term in this context. We should avoid to embrace a – so-to-speak – «organization mysticism».[28]

What aspects of organization should we focus on, so as to provide the most adequate and most tractable ones for explanatory purposes? From a mechanistic perspective, organization has been defined as the «relation between a mechanism as a whole and its components», by virtue of which they «work together to do something».[29] The three dimensions of dependence exhibited by the forms of organization that Kuorikoski and Ylikoski introduced (diversity in the kinds of components, the network structure between the components, and diversity in the kinds of relations) make sense in the cases addressed here. However, an understanding of behaviour in terms of properties of the whole and its constituent parts does not seem to provide sufficiently significant explanatory clues – thus challenging the idea that a mechanistic approach to mental disorders is (always) the road to choose. The following issue is thorny: if we take it that lower levels are constitutive of the whole and therefore cannot be manipulated independently of higher levels, and that both the parts and the whole they constitute are neither independent existences nor related by causal relations, we are left with the problem of accounting for the differences in cognitive outcomes despite (at least apparently) very similar underlying levels. Explanatory progress in mechanistic terms would occur if, for instance, they shed light on the specific features, details and constraints of material realization that affect overall cognitive performance, given that (apparently) analogous levels are actually associated with very different behaviours. In principle, «by finding the basic constituents of organization, we find the things that, if changed, would lead to changes in the property of the whole», while «the properties of relations explain system-level behaviour if we can link changes in the specific properties of such relations [...] to specific changes in the system-level property».[30] These attempts to clarify organization are challenged by the cases described, which seem to indicate that in fact the specific arrangement of constituents does not matter – or does not matter enough – to affect system-level behaviour, and cannot hence provide the best grounding for explanatory practices.

Considering what relations are explanatorily relevant also requires taking a stance on the *purpose* of the explanation itself. As mentioned above, the interpretation and modelling of clinical situations in which cognitive reserve and psychiatric resilience play – or can play – a role are problematic due to difficulties in producing accounts that are both accurate – in the sense of considering all the relevant factors and their mutual relations – and tractable – that is, most effectively employed in practice. Explaining mental disorders also has to do with establishing what elements are doing the work we are most interested in *for clinical purposes*. For instance, how do accommodation/compensation strategies affect the stability/instability of the system at stake (i.e. resulting in standard cognitive performance vs. disorder) so as to account for clinical conditions? Finally, which of the identified interactions between factors involved are the best targets for interventions that can improve health conditions under the circumstances? Addressing organization as resulting in resilient behaviours raises clinical issues, by directly affecting our decisions on whether to actively intervene or not in the functioning of the system at different levels: when and to what extent should

clinical interventions be performed, or might resilience itself provide sufficient mitigation?

In discussing organization, and its explanatory role in mechanistic accounts, Kuorikoski and Ylikoski state that «the determination relation between the properties of the parts and the whole is not a process in time».[31] However, time and order seem to be highly relevant in the cases under consideration. One striking feature that emerges in some studies is the differential functioning of what appear to be resilience-increasing factors in initial and subsequent stages of the disorder. For instance, it has been shown that, in cases of cognitive decline, high education levels may initially constitute a protective factor but lead to accelerated decline after disease onset. Similarly, some longitudinal studies have shown that more years of education and higher premorbid IQ correlate with the later onset of dementia symptoms; but after onset, cognitive decline is actually faster in patients with higher indices of cognitive reserve.[32] So, not only do cognitive reserve factors change the behaviour of the system, these factors also themselves change in relations to changes in the functioning of the system over time. They can play both a functional role with respect to maintaining the normal organization of the system, and a dysfunctional role – as if, as has been hypothesized, at some point the increasing neuropathological load eventually overrides their protective effects.

Studies thus suggest that resilience can give us hints as to how extensive a disturbance a system can withstand before undergoing a significant breakdown of its standard functioning. Is there a clear threshold that is eventually reached, finally cancelling the mismatch between expected cognitive performance and actual cognitive performance? Is it possible to identify breakdown thresholds at which conditions shift from non-pathological to pathological? Establishing thresholds for the benefits of protective factors would allow us to optimize interventions or allow us to forego them if unnecessary or avoidable, from a perspective that would promote a strong focus on the capacity to resist disease and the influence of life-long, distal and sometimes remote factors. Despite the presence in the literature of different operational definitions of resilience, a general tenet that can be inferred from various applications of the concept is that resilience built earlier in life can act as a buffer against the harmful effects of future stressors and/or adverse effects, with different individuals exhibiting different thresholds in response to ongoing daily stressors.[33] Studies on resilience and cognitive reserve should provide hints as to what really holds the pieces together and is responsible for the behaviour of the system, defined as *a set of interdependent components characterized by system-wide responses* – which might not be most effectively expressed in mechanistic explanatory terms.

Finally, as briefly anticipated above, let us stress how discourses on cognitive reserve and resilience also affect diagnosis. If cognitive performance is not what would be expected, what should be taken as the hallmark of the pathology? Should we regard underlying dysfunction or higher-level cognitive performance as the determining factor? *Which* "orchestrated functioning" should we consider when diagnosing? If the focus is on cognitive performance which can actually be observed, then we will not label the person under consideration as diseased and diagnosis will be delayed (given that reserve and resilience are temporary buffers). If, instead, we focus on the underlying neurobiological conditions, the person will be labelled as diseased. Different answers to this question thus lead to a condition being assessed as pathological or not, with clear clinical implications.

In his discussion of complex systems, William Wimsatt has stressed that we need to decide which are the *relevant* components and levels with respect to the *epistemic aim* at stake. Plus, «we need to know more generally how we should order and relate different descriptions of the behaviour of a system, particularly partial descriptions, to construct

explanatory accounts of its behaviour».[34] In the recent philosophical debate on mechanistic explanations, organizational explanations have been recruited to trace dependencies between organizational patterns and system-level properties. It might be difficult not only to decipher whether one has accurately captured some, or possibly all, of the relevant relations, but also whether, in doing so, one has grasped the *tractable* ones. As has emerged from the above reflections, research on cognitive reserve and psychiatric resilience bring important issues to the fore. It encourages us to reflect on what kind of knowledge would enable us to draw inferences regarding the different consequences of analogous systems involving similar constituents, and the capacity of such systems to change and resist change.

4 Concluding remarks

In the sections above, we have shown that resilience presents challenges with respect to a mechanistic account of organization. This mechanistic account – and the crucial explanatory role it has been granted in recent mechanistic theories – does not seem the most effective epistemological tool to clarify what elements should be counted as "responsible" for the system's behaviour in cases of cognitive reserve or psychiatric resilience. This might effectively impact explanatory attempts to identify stable patterns underlying disorders. The challenge is related to the *choice* of *what exactly* organization has to specify to be considered genuinely explanatorily significant. It appears that accommodation strategies and resilient behaviour can hardly be represented in terms of discrete entities, their mutual local interactions, and constitutive relations.

It is a fundamental tenet of the mechanistic framework that the organized activity of the constituent parts of a mechanism produce the very behaviour that characterises the target phenomenon, and that a mechanistic explanation should describe the behaviour and organization of certain constituent entities. In the cases presented above, issues emerged as to how the organisation of the lower-level parts is exactly responsible for the global behaviour of the system. While I am overall well disposed towards mechanistic accounts, I have here stressed cases which suggest the need for correctives. Reserve and resilience shed some doubt on the adequacy and autonomy of mechanistic views for models of cognition and cognitive impairments, explanatory purposes and encourage further epistemological debate along various lines. Let us conclude by mentioning some of these.

To start with, resilience and reserve are usually presented as positive factors, but do they actually and necessarily provide an *epistemic advantage* given that they might make the disorder more difficult to detect? Or do they mask rather than shed light on the relevant causal relations? One goal of explaining is to parse the network of interactions and pick out the most relevant relations – relations that might then also increase our power to predict and intervene effectively. Does taking resilience into account always provide an explanatory advantage (i.e. allow for a better understanding of the system's actual functioning), or can the resulting systemic rearrangement in some cases mask explanatorily relevant underlying causal relations? Does it hinder rather than facilitate comprehension, from a strictly epistemological point of view? Seeing no effect of a given variable doesn't mean that the variable isn't contributing to the behaviour of the system: other parts of the system could be compensating. In this sense, resilience can actually hamper the unravelling of underlying causal relations, temporarily masking important factors.

Does psychiatric resilience work as a unifying concept? On the one hand, the situation described is common to many patients; on the other, as already stressed, remarkable individual differences are observed. Studying phenomena currently classified as cognitive reserve and resilience can provide hints on underlying common systems that not only take proximal but also distal and remote con-

tributing – or counteracting – factors into account from a life-long perspective. For instance, Watson and Joyce claim that «an understanding of whether there is plasticity in the fronto-parietal system that can be modified by life experience would illuminate approaches to the cognitive remediation of both schizophrenia and dementia and have implications for health maintenance in the general public»,[35] thus suggesting that transdiagnostic conclusions could be drawn from the maintenance of functionality. At the same time, exactly what resilient behaviour stands for – i.e. whether for a healthy or pathological condition, whether for a normal or an aberrant response – remains controversial. Rutter, for instance, has suggested that depression itself is essentially an adaptation to adverse conditions, and, in this sense, a sign of resilience, rather than lack of resilience.[36] Clearly, open questions remain here too, in relation to the comprehensibility and manageability of adverse events.

Finally, do cognitive reserve and psychiatric resilience bring some prescriptive, rather than merely descriptive, stance with them? Given that resilience suggests a "better than expected outcome", is it a normative-flavoured concept, one that leads to a conversation about priorities (especially in the case of mental disorders, where socially acceptable/unacceptable behaviours are at issue)? Is resilience therefore desirable, and should we think of resilience as a good thing to be promoted and enhanced?[37] Answers to these questions and reflections on the possible prescriptive meaning of the notion will, again, also affect organizational explanatory accounts, the ways in which the boundaries of the system are defined, and the global behaviour to be pursued among multiple possible equilibria. Questions about the "resilience *of what*" – the exact boundaries of the multilevel and multiscale resistance to change we are interested in – must then be accompanied by reflections on "resilience *for whom*" – discussing, especially in medical contexts, what the target functioning of the system might be. Hence investigations should be pursued to establish whether re-organization in resilience/reserve cases means the ability to return – or strive to return – to some previous, pre-disturbance state, or, rather, to adjust and promote a new functional state. Without denying the importance and explanatory usefulness of the notion of organization, all these reflections are thus meant to stress how, in psychological and psychiatric contexts, reserve and resilience, and the many different paths to resilient outcomes, encourage a debate which could touch upon organizational mechanistic explanations, as well as definitions of disorder, diagnoses and therapeutic choices.

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Notes

[1] J. WAKEFIELD, *The concept of mental disorder. On the boundary between biological facts and social values*, in: «American Psychologist», vol. XLVII, n. 3, 1992, pp. 373-388, here p. 373.

[2] D. MURPHY, *Psychiatry and the concept of disease as pathology*, in: M. BROOME, L. BORTOLOTTI (eds.), *Psychiatry as cognitive neuroscience: Philosophical perspectives*, Oxford University Press, Oxford 2009, pp. 103-117, here p. 103 and p. 113.

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[10] Cf. S. GLENNAN, *The new mechanical philosophy*, Oxford University Press, Oxford, 2017. See also P. ILLARI, J. WILLIAMSON, *What is a mechanism? Thinking about mechanisms across the sciences*, in: «European Journal for Philosophy of Science», vol. II, n. 1, 2012, pp. 119-135; D. KAPLAN, C. CRAVER, *The explanatory force of dynamical and mathematical models in neuroscience: a mechanist perspective*, in: «Philosophy of Science», vol. LXXVIII, n. 4, 2011, pp. 601-627.

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[12] J. KUORIKOSKI, P. YLIKOSKI, *How organization explains*, in: V. KARAKOSTAS, D. DIEKS (eds.), *EPSA11 Perspective and foundational problems in philosophy of science*, Springer, Dordrecht 2013, pp. 69-80, here p. 69.

[13] C. CRAVER, *Explaining the brain*, Oxford University Press, Oxford, 2007, p. 189.

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[16] Cf. S. CHANRAUD, A.-L. PITEL, E.M. MÜLLER-OEHRING, A. PFEFFERBAUM, E.V. SULLIVAN, *Remapping the brain to compensate for impairment in recovering alcoholics*, in: «Cerebral Cortex», vol. XXIII, n. 1, 2013, pp. 97-104.

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[18] D. BARTRES-FAZ, E.M. ARENAZA-URQUIJO, *Structural and functional imaging correlates of cognitive and brain reserve hypotheses in healthy and pathological ageing*, in: «Brain Topography», vol. XXIV, n. 3-4, 2011, pp. 340-357, here p. 340 - italics added.

[19] For attempts to measure cognitive reserve, see e.g. B.R. REED, D. MUNGAS, S.T. FARIAS, D. HARVEY, L. BECKETT, K. WIDAMAN, L. HINTON, C. DECARLI, *Measuring cognitive reserve based on the decomposition of episodic memory variance*, in: «Brain», vol. CXXXIII, Pt. 8, 2010, pp. 2196-2209; L.B. ZAHODNE, J.J. MANLY, A.M. BRICKMAN, K.L. SIEDLECKI, C. DECARLI, Y. STERN, *Quantifying cognitive reserve in older adults by decomposing episodic memory variance: Replication and extension*, in: «Journal of the International Neuropsychological Society», vol. XIX, n. 8, 2013, pp. 854-862.

[20] It has been shown that when part of the fron-

to-parietal system is damaged, an increase in activity occurs throughout the system. This could either reflect the use of different strategies or the recruitment of more cortex to compensate. See A. WOOLGAR, D. BOR, J. DUNCAN, *Global increase in task-related fronto-parietal activity after focal frontal lobe lesion*, in: «Journal of Cognitive Neuroscience», vol. XXV, n. 9, 2013, pp. 1542-1552.

[21] Cf. M.J. LIND, R.C. BROWN, C.M. SHEERIN, T.P. YORK, J.M. MYERS, K.S. KENDLER, A.B. AMSTADTER, *Does parenting influence the enduring impact of severe childhood sexual abuse on psychiatric resilience in adulthood?*, in: «Child Psychiatry & Human Development», vol. XLIX, n. 1, 2018, pp. 33-41. Among the first relevant works on psychiatric resilience, see M. RUTTER, *Resilience in the face of adversity: Protective factors and resistance to psychiatric disorders*, in: «The British Journal of Psychiatry», vol. CXLVII, 1985, pp. 598-611. On psychiatric resilience being something different from the mere absence of disease, see A.M. ALMEDOM, D. GLANDON, *Resilience is not the absence of PTSD any more than health is the absence of disease*, in: «Journal of Loss and Trauma», vol. XII, n. 2, 2007, pp. 127-143.

[22] Cf., e.g., S. COLLISHAW, G. HAMMERTON, L. MAHEDY, R. SELLERS, M.J. OWEN, N. CRADDOCK, A.K. TAPAR, G.T. HAROLD, F. RICE, A. THAPAR *Mental health resilience in the adolescent offspring of parents with depression: a prospective longitudinal study*, in: «Lancet Psychiatry», vol. III, n. 1, 2016, pp. 49-57) consider high-risk adolescents (e.g. given severe parental depression) and show that child, family, social, and lifestyle factors together positively contribute to adolescent mental health resilience.

[23] Cf. E.C. LONG, S.L. LÖNN, J. LI, P. LICTHTENSTEIN, J. SUNDQVIST, K. SUNDQVIST, K.S. KENDLER, *Resilience and risk for alcohol use disorders: a Swedish twin study*, in: «Alcoholism. Clinical & Experimental Research», vol. XLI, n. 1, 2017, pp. 149-155; S.R. HORN, D.S. CHARNEY, A. FEDER, *Understanding resilience: New approaches for preventing and treating PTSD*, in: «Experimental Neurology», vol. CCLXXXIV, Pt. B, 2016, pp. 119-132. On genetic and environmental factors, and attempts to elaborate an integrative framework, see e.g. K.W. CHOI, M.B. STEIN, E.C. DUNN, K.C. KOENEN, J.W. SMOLLER, *Genomics and psychological resilience: A research agenda*, in «Molecular Psychiatry», vol. XXIV, n. 12, 2019, pp. 1770-1778.

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[25] Cf. A. WATSON, E. JOYCE, *Cognitive reserve and neuropsychiatric disorders*, in: «Current Opinion in Behavioral Sciences», vol. IV, 2015, pp. 142-146.

[26] A. SHRIVASTAVA, A. DE SOUSA, P. LODHA, *Resilience as a psychopathological construct for psychiatric disorders*, in: Y.K. KIM (ed.) *Frontiers in psychiatry. Artificial intelligence, precision medicine, and other paradigm shifts*, Springer, Singapore 2019, pp. 479-489, here p. 479.

[27] Cf. A. LEVY, W. BECHTEL, *Abstraction and the organization of mechanisms*, in: «Philosophy of Science», vol. LXXX, n. 2, 2013, pp. 241-261

[28] Y. KUORIKOSKI, P. YLIKOSKI, *How organization explains*, cit., p. 70.

[29] Cf. B. CLARKE, D. GILLIES, P. ILLARI, F. RUSSO, J. WILLIAMSON, *Mechanisms and the evidence hierarchy*, in: «Topoi», vol. XXXIII, n. 2, 2014, pp. 339-360.

[30] Y. KUORIKOSKI, P. YLIKOSKI, *How organization explains*, cit., pp. 74-75.

[31] *Ibid.*, p. 71.

[32] Cf., e.g., A. SOLDAN, C. PETTIGREW, S. LI, M.C. WANG, A. MOGHEKAR, O.A. SELNES, M. ALBERT, R. O'BRIEN, BIOCARD RESEARCH TEAM *Relationship of cognitive reserve and cerebrospinal fluid biomarkers to the emergence of clinical symptoms in preclinical Alzheimer's disease*, in: «Neurobiology of Aging», vol. XXXIV, n. 12, 2013, pp. 2827-2834; S.R. RAPP, M.A. ESPELAND, J.E. MANSON, S.M. RESNICK, N.R. BRYAN, S. SMOLLER, L.H. COKER, L.S. PHILLIPS, M.L. STEFANICK, G.E. SARTO, WOMEN'S HEALTH INITIATIVE MEMORY STUDY, *Educational attainment, MRI changes, and cognitive function in older postmenopausal women from the Women's Health Initiative Memory Study*, in: «International Journal of Psychiatry in Medicine», vol. XLVI, n. 2, 2013, pp. 121-143.

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[35] A. WATSON, E. JOYCE, *Cognitive reserve and neuropsychiatric disorders*, cit., here p. 144.
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