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**Review of the Dissertation**

Author: Kacper Kondrakiewicz
Title: Characterization of central amygdala circuits activated by social transfer of fear
Supervisor: Prof. Ewelina Knapska

In this thesis, Kacper Kondrakiewicz examines the mechanisms through which a rat responds to witnessing another rat receive shocks. In particular, Mr Kondrakiewicz examines the behavior of the rats while witnessing the shocks, and while reactivating or inhibiting neurons in the CeA that were activated while witnessing the shocks to the demonstrator. The main findings are that observers show elevated freezing during shock observation, and that reactivation of the CeA neurons recruited during shock observation later led to the flexible recruitment of freezing if in a small environment, and avoidance when the environment allows for hiding. Additionally, although demonstrators that froze more triggered higher levels of freezing in the observers, the relative timing of freezing in observers and demonstrators was not significantly linked. Together with the flexible recruitment of freezing vs hiding during reactivation of the neurons, this suggests that the transmission across the animals is mediate by emotional contagion rather than simply motor mimicry. Together, these main experiments provide two significant conceptual advances: they establish the sufficiency of CeA neurons recruited during shock observation in triggering flexible nocifensive reactions and they help distinguish that fear transmission is more likely to be a form of emotional contagion rather than mimicry. Importantly, the introduction and the discussion section of the thesis elegantly and maturely identify these conceptual advances and situate them accurately in the scientific landscape. Based on these main experiments alone, and the introduction and conclusions, I find that the candidate has proven his ability to perform scientific research.

A number of additional experiments are reported in the thesis, and contribute to the richness of the work.

Foremost, in an interesting experiment, the candidate precedes or follows the shocks to the demonstrator with a tone. Results show that observers learn to fear the tone in both cases, as shown by freezing on the next day. However, there is no clear distinction in freezing level across these observers. This is an intriguing finding, that is interpreted in the discussion, in a convincing fashion, as suggesting that the timing of the internal state triggered by shock observation is perhaps not 'crisp' enough to trigger different learning based on the important difference in contingency.

Second, the candidate has performed a comparatively smaller-scale study on connectivity that represents an interesting approach to further understanding what characterize CeA cells responding during the observation of other animals receiving shocks. Unfortunatly, the sample size used in the tracing experiments (n=3 observers and n=4 controls) did not allow for robust statistical analysis. Although this data suggests a special role of BLA->CeA and ACC->CeA in terms of co-labelling of retrograde tracing and cFOS, the sample size was underpowered. It is unfortunate, that this intriguing data has not been expanded in sample size to provide more robust data, but the candidate does discuss the results with appropriate care. Also the Chemogenetic manipulation of the BLA->CeA connections, provides intriguing trends visible for avoidance and distance travelled, that are suggestive but not quite conclusive despite sample sizes that are in accordance with the standards in the literature.

In summary, this thesis presents work of high scientific quality, that addresses important questions, and provides significant conceptual advances. In particular, I was compelled by how maturely the candidate reviews the state of the literature, identified the key issues worth exploring further, and interprets the results keenly in how they bring our understanding of these key issues forwards. Through this thesis, the candidate demonstrates both his experimental and scholarly aptitude for cutting edge research. I therefore enthusiastically recommend proceeding to the doctoral defense of the thesis.

Minor comments

- I generally gound that the introduction (section 1) was unusually well written, and provided a very balanced introduction of high scholarly quality of the key concepts.

-p32: I couldn't quite find a specification of the sex of the animals. Were only male or female rats used? Or both? And if both, did you notice or consider sex-differences?
P33: Paradigm: I couldn't quite identify if the fear contagion paradigm was conducted under darkness, or whether there was sufficient light for the observers to see the demonstrator. This is relevant for the modalities that might be responsible for the communication, and this may influence the timing of the vicarious-responses and therefore how strong the temporal association between observer and demonstrator behavior was and how strong the dissociation between cs-first and cs-second can be.

-p56 'auditory cue proceeded' should read 'auditory cue preceeded'

-p69 'They indicated that behavioral mimicry – which seems to be favored by shared circuit approaches'. This sentence, and some of the other discussion seems to suggest that shared circuit approaches think that the connection between individuals is mainly motor. I understand where that stems from – in that mirror neurons were originally discovered in the motor system, which could support mimicry. However, my feeling as one of the early promotor of the shared circuit approach, is that very early already, the idea was that shared circuits in the motor system could support the matching of actions, and shared circuits in the 'limbic' system (insula, acc, amygdala) could support emotional contagion. Hence, I applaud the thesis in the way it distinguishes emotional contagion and mimicry, and provides support that vicarious freezing is not mimicry but rather emotional contagion. I just thought wasn't sure that share circuit proponents favour mimicry in general, and personally, at least, do not…

-p70. 'These results are consistent with the hypothesis that during fear contagion behavior of observers is driven by signals which are poorly specified in time'. It might be interesting to discuss whether we might expect this to be different if observers would have been pre-exposed to shocks?

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I sometimes have the intuition (as yet untested) that naïve observers rely on more innate signals (incl olfaction), while pre-exposed observers can utilize acquired cues (see the work of Moita), that might provide temporally more precise information?

P71. 'vsery high expression' should read 'very'.

Yours sincerely

Prof Christian Keysers, PhD
Amsterdam, 11 March 2021

Champalimaud
Foundation

Lisbon, April 12, 2021

**Review of the PhD thesis by Kacper Kondrakiewicz, entitled "Characterization of central amygdala circuits activated by social transfer of fear"**

This thesis addresses an important and timely question that regards the neuronal mechanisms that mediate social transfer of fear, or emotional fear contagion. The spread of defensive responses across individuals in a group is a widely reported phenomenon, however the underlying neuronal mechanisms are still poorly understood. In his thesis Kacper Kondrakiewicz sets out to test the shared circuits hypothesis, according to which neuronal circuits of vicarious emotions, that is triggered by emotional contagion, partially overlap with circuits involved in similar emotional caused by first-hand experience. To this end he chose to focus on the central amygdala, a structure critically implicated in the modulation and expression of defensive responses and its inputs. Importantly, the microcircuit of CeA is characterized to some extent, making this structure an ideal starting point. Kacper Kondrakiewicz performed challenging experiments that combined various complementary approaches, from the use of several behavioral paradigms, through immunohistochemistry, to opto- and chemogenetics. First, Kacper Kondrakiewicz examined whether fear contagion resulted from behavior copying, which could possibly explain the use of a shared circuit and found that was not the case. Still, optogenetic manipulations targeting cells activated by vicarious fear demonstrated a role of central amygdala (CeA) in this process, thus demonstrating that the CeA is involved in both vicarious and first-hand driven fear. Experiments aimed at determining which sub-population of CeA neurons were unfortunately not conclusive. Finally, Kacper Kondrakiewicz looked into the inputs of CeA that might be particularly activated by vicarious fear. Although the sample size was small, these experiments lead to interesting observations, namely that CeA projecting neurons from the basolateral amygdala and the anterior cingulate cortex might be important in driving vicarious fear and that projections typically involved in first-hand experienced fear, such as the prelimbic cortex are inhibited.

The thesis is well written and scholarly, and the experiments challenging and elegantly designed.

Please find below my comments. They are meant to promote an interesting discussion. All suggestions for experiments, should be viewed as thought experiments, not as requirements for the thesis.


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**Introduction**

The introduction is well, structured, written and clear.

1) Theoretical framework:

The introduction starts by putting the subject of this thesis, the mechanism of social transfer of fear, in the broader context of emotional contagion and mimicry. The concept of the Russian doll places mimicry as the simplest mechanism that could drive emotional contagion. The idea of a crescendo of complexity might not be so very useful. For example, in the context of social learning, mimicry is considered one of the most complex forms, being simpler forms of social learning, emulation, stimulus enhancement etc. How would these fit in the Russian doll it not very clear. It could be interesting to discuss a bit more how the use of social information and learning have evolved.

2) Defensive behaviors.

Although not central to the current thesis project, in this section a dichotomy between passive and active defensive responses is put forth. I disagree with this categorization, as freezing, although characterized by immobility is unlikely to be a passive behavior. Could this have any implication regarding emotional contagion, so far mostly demonstrated using freezing as a behavioral read-out?

3) Microcircuit of CeA and it's functional role.
   i) The description of the CeA's microcircuitry is complete and up-to-data. A diagram, maybe for the presentation at the thesis discussion, would be extremely helpful. It is not clear to me how do the different markers of opposing cells (CRF+/-, SOM +/-, PKC+/-) overlap and the numbers of each sub-population. If these numbers exist, they could be very useful for the discussion (see comment below in the results section).
   ii) The discussion on the behavioral functions of the CeA is quite interesting. Although for a long time the CeA was seen as a relay station, a view championed by Joseph LeDoux. He was also the first to establish a role for this structure in learning (Wilensky et al). This study is referenced later on, still it is very relevant in this section (2nd paragraph page 20). Maybe here a diagram such as the one in the review article by fadok et al (2018) would be very useful.

4) Circuits of fear contagion
   i) In the section on behavioral paradigms of fear contagion it is stated that "when tested alone 24 hours later, the observers still react to the auditory cue with freezing (Cruz et al., 2020; Pereira et al., 2012)". However, in these studies, observer rats were never tested to the auditory cue used to condition the demonstrators. They were tested to the context in which they were shock to test whether they learned from their prior self-experience with shock.
   ii) On page 24, 3rd paragraph it is not clear what is meant by "Recently it was shown, with combination of electrophysiological and optogenetic techniques, that the projections from ACC to BLA are preferentially activated by auditory cues during observational conditioning (Allsop et al., 2018)". It may be less clear to me, since we have demonstrated a role of auditory cues in social transmission of fear, but in this study the auditory cue to which cells


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respond is the auditory stimulus to which the demonstrator is conditioned. This should be made more explicit.

5) At the end of the introduction there is a very nice discussion regarding strengths and weakness of the various techniques used in the thesis. I think to would be nice and relevant, given the results presented in this thesis, to discuss how to interpret artificial gain and loss of function manipulations to establish causal relationships.

**Methods:**

The results section is detailed and clear.

I have a couple of questions:

1) It was not very clear whether, in the optogenetic experiments, the same or independent groups of animals were tested in the exploration, social interaction and recall tests. If the same animals were tested, what was the order in which they were tested? Was this counterbalanced?
2) Social interaction - Were there any agonistic interactions? If yes, it would have been interesting to quantify those. The fear contagion session might have impacted the social hierarchy in the dyads.

**Results:**

The results are very clearly laid out. Although the behavioral analysis on synchrony is very nice and extensive, I feel that given the richness of the data sets, from the various experiments run, the data could have been explored a bit further. For example, it would be interesting to see: timelines of the behaviors measured, examples of trajectories, or more importantly analyzes of the various 'avoidance' behaviors, separately.

It would be helpful, for the presentation to have the timeline of the experiment next to the results of each experiment.

1) Fear contagion paradigm
   i) Observer rats show robust vicarious freezing and decreased exploratory behaviors. These are naïve observer rats. As mentioned in the discussion, this finding is at odds with other studies. One possible explanation is the use of more shocks. Therefore, it would be interesting to see the behavioral changes over the course of the fear contagion session, since it might elucidate the reason for this discrepancy relative to other studies.
   ii) It is not clear what each datapoint in figure 7 represents – is it summed freezing over the whole test session, only once the demonstrator started receiving shocks (after the baseline period), the average freezing per animal?
   iii) Figure 8 shows overlapping USVs, one affiliative and one alarm call, indicating that the two rats are vocalizing simultaneously. It is not clear to me how it implies that observers also emit alarm calls. It is possible that the observer only emitted affiliative calls and all alarm


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calls were emitted by the demonstrators. Overlapping alarm calls are probably very difficult, if at all possible, to detect.

iv) Figure 13 and several figures thereafter have a typo in the plot's legend: 'frist' instead of first.

2) Fear learning through observation

These are very interesting results (see section regarding the discussion).

i) Given that all rats froze robustly in both groups, additional controls could be interesting. One could try to test if observing the demonstrator receive shocks is necessary, or whether the display of defensive behaviors by the demonstrator suffices to induce observational fear learning. This would be an interesting point of discussion.

ii) Figure 15 shows a small but consistent trend for more freezing in CS first. Would the summed freezing during the whole test session be different across the two groups?

3) Activation of CeA neurons

i) No difference was found between activation with fear contagion versus controls, although both groups showed increased activation relative to home cage controls. Still there was a small trend towards more activity in the fear contagion group. This analysis was performed on the whole CeA, could it be that if one would restrict the analyses to specific sub-regions, for example CeL or CeM, or even anterior versus posterior regions of CeA, would show interesting differences?

ii) When specific cell types were analyzed for their activation, using double labelling, again no significant differences were found. The analysis examined whether in the c-fos positive pool of cells, there was enrichment of a particular cell type. Maybe it should be considered as a complement the following analysis: the fraction of CRF or PKCg that are c-fos positive. If the relative size of the cell populations is very different than this analysis might yield a different result. It could be that a subpopulation that is small, is however very much activated by vicarious shock.

4) Optogenetic manipulation

Activation of c-fos labeled cells (tagged during fear contagion) during the exploration test yielded very interesting results. Rats spent more time in avoidance behavior and explored less the large arena. In addition, when tested in the small chamber used for fear contagion, rats froze more.

i) Although inactivating the c-fos tagged cells did not affect avoidance and exploratory behaviors, it did increase the distance travelled. During the activation experiment (Fig 18) the distance traveled showed a similar trend, albeit not statistically significant. It would be interesting to examine the trajectories to see whether there are qualitative differences between the rats that had the tagged cells activated or inhibited. Finally, it would have been interesting to whether inhibition would decrease freezing in the context where fear contagion took place.


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5) Functional tracing
Although the sample size was small, this experiment yielded some surprising results, namely the decreased activity of specific inputs to CeA, where one would expect to see an increase, namely in the pre-limbic cortex, CA1 and anterior insular cortex. It would have been interesting to see how home cage controls would look like.

**Discussion**

With his thesis work, Kacper Kondrakiewicz convincingly demonstrates that rats can display fear contagion, even in the absence of prior experience with threat, that they learn from it and that it is unlikely to constitute behavioral mimicry. In addition, this body of work nicely shows that the CeA, involved in the modulation and triggering of defensive states by first-hand experience with threat, is also involved in fear contagion. These findings are consistent with the shared circuits framework. It does not however provide a definitive answer regarding a more fine-grained analysis, that is, whether first-hand and vicarious fear rely on the same populations of cells within this structure. I have a few comments regarding the discussion.

1) Role of prior experience with first-hand experienced threat. Differences in protocol have bene proposed. In addition to the intensity of the threat the demonstrator is subjected to, I wonder if observation of an immediate versus a distant threat might contribute to the discrepancy. In addition, there might be known differences, from comparative studies on fear and anxiety between strains, that could explain the discrepancies. Are Wistars typically more anxious/fearful?

2) Behavioral mimicry. It would be interesting to discuss at the defense alternative explanations for the lack of tight temporal correlations in freezing between rats. My lab has previously demonstrated a role of freezing, detected through the onset of silence, as the cue that mediates social transfer of fear. Could it explain the lack of close temporal coupling? If so, how?

3) Observer rats showed a small amount of learning. One possibility is that the US is weak, as it is vicarious in nature. However, the demonstrator starts freezing upon the first few shocks and probably stays freezing through, as the pre-CS freezing suggests (would be nice to see the time course of freezing a suggested above). Could the sustained freezing regardless of the CS degrade the contingency for the observer? Or is the time-locked shock response the trigger. The fact that the temporal relationship between the CS and the US does not matter for the observer, could be a result of the weak differences in freezing by the demonstrator across the two training protocols. It could also be second order conditioning through contextual learning. It would be very interesting to discuss these alternative explanations.

4) It would be interesting to discuss, alternative methods to single-unit recordings that allow both following the same cell during vicarious or first-hand fear while keeping a handle on cell type, and how such experiments would contribute to our understanding of CeA's function. Putting it in an even broader sense, how does it contribute to our understanding of emotions.


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5) The rational for citing these particular papers as evidence for lack of ability to learn though observation is unclear to me.
“No mimicry detected in the fear contagion paradigm could suggest that the naïve rats, although reacted with freezing to aversive signals from the demonstrators, were not able to learn through observation (Allsop et al., 2018; Atsak et al., 2011; Cruz et al., 2020; Han et al., 2019).”

The work expanded in this thesis is original, addressing the timely issue of the mechanisms underlying emotional contagion. The experiments performed are well founded. The results presented in this work is interesting and timely and will likely result in a good publication. Although some experiments did not yield conclusive results, they raise interesting questions.

For these reasons I believe meets the requirements to be discussed publicly.

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HR EXCELLENCE IN RESEARCH

**Review of the PhD thesis of Kacper Kondrakiewicz, MSc:**

**„ Characterization of central amygdala circuits activated by social transfer of fear".**

Presented thesis is focused on a very interesting and important scientific problem, namely on mechanisms underlying social transfer of emotion, especially fear. Data analyzed in this dissertation are based on experiments carried out in the Laboratory of Emotions Neurobiology at the Nencki Institute of Experimental Biology in Warsaw. The experimental approach and thesis preparation were supervised by Professor Ewelina Knapska.

The thesis has been prepared in English as a standard monograph and comprises 100 pages with typical chapters and sections.

The research project designed as the basis for this dissertation aims to shed light on two main questions:

- What is the main essence of the emotion transfer
- What are the neuronal circuits responsible for this phenomenon

"Introduction" is the first chapter of the monograph summarizing actual facts and hypotheses relating to the social transfer of emotion – emotional contagion. All key methods used in the experimental part of the thesis are also listed and clarified in this chapter.

Significant part of "Introduction" is focused on the differences between emotional contagion and behavioral mimicry. The author indicates many examples coming from other species than mice and rats. Numerous citations from the bibliography part support opinions and statements presented in this chapter. The phenomenon of motor mimicry has been introduced and explained as form of simple copying of behavior of other individuals. It has to be mentioned however, that the mimicry can apply not only to behavioral level, but also to physiology or morphology.

A good example are coral reef fish species, which mimics the cleaner fish behaviorally and morphologically to benefit from the same food source. This kind of behavior is also very important for species maintenance strategy and is not only a useless behavioral imitation like Zelig from the Woody Allen's film.

As I have already mentioned above, in the chapter "Introduction" there are three sections devoted to methods utilized in the thesis. This is a good opportunity to shed some light on the sophisticated methods not commonly used in behavioral studies.

The next chapter are "Research aims", where the hypotheses and the basic research aims are pointed out.

- The first hypothesis says: in case of emotional contagion the freezing behavior of the observer can be predicted as the consequence of the demonstrators freezing behavior.
- The next one says: the neurons of the central amygdala circuits, which are being activated in observer, are similar to the neurons being activated while fear conditioning.

In the third chapter "Methods", the author presents in detail all methods applied in the thesis, including experimental animals, behavioral testing, optogenetics and statistics. For me it is not clear why the USV recording methods are not reviewed in section 3.2 "Behavioral testing", but in section 3.10. In the section 3.1 "Animals" a more detailed information on the Wistar strain is also missing. On the pages 41 and 42 in the chapter 3 are placed figures 3 and 4 illustrating the expression of vectors utilized in the optogenetic and chemogenetic experiments. In these figures scale bars are missing. An important part of the chapter 3 is also the scheme summarizing the whole experimental design and the table indicating the number of animals tested in all procedures. At this point however I would like to ask, about the small numbers of animals utilized in "optogenetic – fear recall, ChR2" and "functional tracing" approaches? Is it possible, that the small animal number has affected the statistical reliability of obtained results?

The fourth chapter "Results" is a broad and detailed presentation of the thesis results. Sections 4.1 – 4.4 deals with the behavioral part of the study utilizing animals not modified by the chemogenetic or optogenetic manipulations. Behavioral analysis has been focused on the freezing behavior, rearing, and walked distance. I have however a problem with the precise definitions of freezing behavior and walking in the cage. Let's imagine, that the test lasts 12 minutes and during this time the animal is not moving (freezing) or is moving (walking). What is the animal doing in the rest of time, if (see the graph) the sum of rest time and walking time is not equal to 12 minutes? If the third activity is rearing the sum is still not 12 minutes. For more clarity it might be better to use distance as the measure of "walking" or instead of the term "walking" use the term "moving time" using then time units.

The results of USV recordings and analysis are summarized in the section 4.2 "Ultrasonic vocalizations". After the detailed lecture of this section some comments seem to be necessary. Firstly, either in "Introduction" or in "Methods" and "Results" there are no comments on the ultrasonic vocalization of rats. It would be important contribution to interpretation of USV results, especially because of the major differences between the USV activity of rats and mice. Secondly, I would like to ask the thesis author for the alternative option to record the USV of demonstrators and observers in separate cages. If we assume, that the emotional contagion depends on the olfactory information (see the authors opinion in the chapter "Discussion"), then the distance of e.g. 1 meter between the demonstrator and observer would be meaningless for emotion transfer but would enable parallel but separate recordings of USV for both animals.

"Discussion" is the next chapter of the thesis. Experimental data are discussed on the basis of the cited literature and authors own considerations. Kacper Kondrakiewicz is emphasizing the fact, that in contrast to many other studies, the observer rats utilized in the study have been never electrically stimulated with a food shock, what is an advantage in this kind of experimental approach. We have to keep in mind, that one of the main goals was to define the neuronal circuits responsible for the emotional contagion.

The principal issue discussed in the section 5.1 is the attempt to define the differences between "fear contagion" and "behavioral mimicry" in other words, what is the difference between emotion transfer and a simple imitation of behavior. In my opinion, this dissertation is an attempt to evaluate both phenomenon in respect to their evolutionary importance. Both of them are directed on survival strategy in case of exposure to danger. The "fear contagion" however, could result in development of more advanced cognitive functions, while "behavioral mimicry" seems to by only simple copying of behavior. The key feature and a basis for discrimination between "fear contagion" and "behavioral mimicry" might be the time lag between the demonstrated behavior and its recapitulation by the observer. Behavior repeating by the observer after a time shift might indicate involvement of memory and learning mechanisms, and hence indicate transfer of emotions rather, than "behavioral mimicry".

In the section 5.2.1. the author deals with interpretation of experiments focused on neuronal circuits involved in fear contagion. These experiments have been based on concomitant expressional analysis of c-Fos and other neuronal markers. At first glance it becomes clear, that methodological aspect was the main issue of this approach. As commonly known, immunohistological staining depends strongly on the repeatability of the staining procedures and in the case of multiple staining, putative flaws might be replicated. As the consequence, the evaluation of staining results is not clear, especially

when the expression of one of the proteins depends on animal behavior. Application of more sophisticated methods like e.g. single-cell RT-PCR would be of advantage.

Another problem visible in the "Discussion" is the lack of similar studies coming from other laboratories, which could be a reasonable material for comparison of own data. Looking at the literature collected in the chapter "Refences" (210 items) we can easily notice, that the vast majority of studies utilized mice and not rats. I can absolutely agree with the statement cited in "Discussion": "Although it is commonly assumed that data from these two model organisms can be used jointly, there are numerous behavioral and neuronal differences between the two species (Ellenbroek & Youn, 2016)". Mouse and rat are very different species, however some scientist consider mouse as a small rat, while they both differ essentially in respect to physiology and behavior. Kacper Kondrakiewicz is awake to this fact, but it makes the interpretation of results not easier.

On the page 71, I have noticed the formulation "passive exploratory strategy". For me this sentence is by itself contradictory. Exploration is an active process of environment investigation, even if it is not necessarily combined with covering of a distance. The author states the following: "The word 'strategy' reflects the fact that stimulating the population did not always evoke one stereotypical behavior, for example freezing. Instead, depending on the testing environment, different responses to the light stimulation could be detected – such as avoidance, decrease of rearing or freezing. To sum up, the 'fear contagion' neurons seem to promote different forms of reducing active environment exploration." For me and obvious conclusion is, that the stimulation of a neuronal population results in a decrease of the exploratory activity. Despite other kinds of behavior observed instead, the lack of exploratory activity is not a passive exploration.

Conclusions are summarized in the section 5.3. From the formal point of view "Conclusions" as a new chapter number 6 would, in my opinion, better suit the dissertation. This part of thesis has not a typical form of statements listed as answers on the hypotheses presented at the beginning of the dissertation. It is not a criticism, because the thesis results do not give simple answers. This section is a summary of the experimental results and a critical interpretation based on the PhD students own considerations. The author is awake about the weaknesses of the experimental part and indicates future research directions utilizing additional and more precise and adequate technics.

To summarize the whole dissertation we can conclude, that the phenomenon of emotional contagion and fear transfer is based not on a simple behavioral imitation and more research is needed to define neuronal circuits responsible for this kind of behavior.

**Summary:**

The reviewed PhD thesis is effect of research based on an up-to-date methodology and is effect of a high-level scientific study.

The dissertation of Kacper Kondrakiewicz has been prepared in accordance with valid regulations applying to PhD thesis in Poland (art. 187 ustawy z dnia 20 lipca 2018 roku, Prawo o szkolnictwie wyższym i nauce (Dz. U. z 2018r. poz. 1668 oraz załącznik nr 1 Regulaminu Rady Naukowej z dnia 13.04.2018 roku) and can be processed in the next steps of the PhD procedure.
In my opinion the PhD thesis of Kacper Kondrakiewicz deserves to be distinguished as an outstanding dissertation.

Przedstawiona rozprawa spełnia wszystkie wymagania określone w art. 187 ustawy z dnia 20 lipca 2018 roku, Prawo o szkolnictwie wyższym i nauce (Dz. U. z 2018r. poz. 1668) oraz załącznika nr 1 Regulaminu Rady Naukowej z 13.04.2018 roku, w związku z czym wnioskuję o dopuszczenie Pana mgr Kacpra Kondrakiewicza do dalszych etapów przewodu doktorskiego.
Jednocześnie wnioskuję o wyróżnienie rozprawy.

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prof. dr hab. n. med. Jarosław Barski