Alterations of attention and impulsivity in the rat following a transgenerational decrease in dietary omega-3 fatty acids

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Abstract

Polyunsaturated fatty acids (PUFAs), particularly the omega-3 PUFAs, are thought to be involved in neuronal processes, to play a role in neurodevelopmental disorders and to be important for the integrity of central nervous system functioning. The present study investigated the effects of nutritional omega-3 PUFAs on attentional functions and impulsive behavior in Wistar rats. For this purpose, female Wistar rats were fed an omega-3 deficient diet over several generations, and the dams of the seventh generation were randomly assigned to two diet groups and fed an omega-3 deficient or an omega-3 sufficient diet. In addition, a group of previously untreated dams was fed an omega-3 sufficient diet. The male offspring of these three diet groups were tested using an established paradigm for the assessment of attention and impulsive behavior, i.e. a modified version of the five-choice-serial-reaction-time task (5CSRTT). The present data show that the deficiency of omega-3 PUFAs over generations led to substantial changes in attentional processes and impulsive behaviors. The impairments associated with an omega-3 deficiency were partly corrected by treatment with the omega-3 sufficient diet in the last generation of the omega-3 deficient group which showed substantial improvements in attention parameters. While there were no significant effects of dietary modifications on psychomotor activity levels, there was some evidence for changes in impulsive behavior. In conclusion, transgenerational dietary changes in the availability of omega-3 PUFAs led to changes in attentional processes and impulsive behavior in rats, supporting the hypothesis that omega-3 PUFAs play a role in cognitive and behavioral processes. The present findings offer a promising approach in the investigation of the role of omega-3 PUFAs in a variety of cognitive and behavioral domains.

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Keywords: Omega-3 fatty acid; Polyunsaturated fatty acids; Attention; Impulsivity; Activity; Rat; Neurodevelopmental disorders; Psychiatric disorders

1. Introduction

Polyunsaturated fatty acids (PUFAs) such as ω-3 fatty acids are known to play an important role in neuronal development and functioning of the central nervous system [1,2]. Long-chain (LC) PUFAs such as eicosapentaenoic acid (EPA, C20:5ω-3), docosahexaenoic acid (DHA, C22:6ω-3) and arachidonic acid (AA, C20:4ω-6) exert an influence on numerous neuronal processes through the regulation of membrane fluidity and by modulating synaptogenesis and neurotrophic factor expression, neurogenesis, and neurotransmission [1–3]. Studies in humans indicate that a deficiency in ω-3 fatty acids leads to an imbalance of the ω-3/ω-6 PUFAs ratio, affects neurocognitive abilities and is associated with developmental disorders [1,2,4]. In this context, a possible impairment in metabolism of PUFAs has been discussed as a potential risk factor for the development of neuropsychiatric disorders such as major depression, bipolar disorder, schizophrenia, Alzheimer’s disease and attention deficit hyperactivity disorder (ADHD) [1,2,5,6].

Furthermore, several studies have shown that an adequate amount of LC-PUFAs is important during prenatal development (particularly in the last trimester of gestation) and after birth [2,3,7–11]. Given the presence of AA and DHA in the breast milk of humans and other placental mammals, the role of these
substances in supporting infant growth and development is clear [12–14]. After birth, the growth of the central nervous system (CNS) rapidly increases, and the brain is therefore sensitive to a deficit of PUFAs [3,15]. An adequate amount of ω-3 and ω-6 PUFAs in breast milk is highly contingent upon quality and composition of fat intake in the mother’s diet [3,16].

Several studies have demonstrated that a lack of DHA in preterm infants through the maternal diet is associated with reduced concentrations of DHA in the cerebral cortex and of DHA-phospholipids in red blood cells [10,17–19]. Additionally, it has been demonstrated that infants fed with a diet low in DHA are at increased risk of developing neurological and neurocognitive problems, e.g. lower IQ scores and visual impairments [20–26]. Moreover, such children have been shown to be at increased risk of dementia and cognitive decline in later life, compared to breast-fed or DHA-fed groups [27–30]. These results show that the infant brain is vulnerable to dietary changes in the amount of PUFAs, and especially to a diet deficient in ω-3 PUFAs [3,16].

These findings indicate a possible link between ω-3 PUFAs and neurodegenerative and neuropsychiatric disorders [1,2,5,6]. For example, in some studies, the symptoms of ADHD have been associated with an ω-3 PUFA deficiency [31]. Other studies found prenatal influences of ω-3 PUFA deficiency on neurocognitive capacity [20]. However, the present state of evidence is far from conclusive. There are substantial differences in methodology, sample size and measured parameters between the available studies [1,3]. Moreover, the evidence for positive effects of ω-3 PUFA supplementation and for negative effects of diets deficient in ω-3 PUFAs is inconclusive [3,32,33]. Since dietary intake is the only source of PUFAs in human beings [1], a supplementation of ω-3 PUFAs can have neuroprotective effects, at least in infants [32,33]. In this context, it is important to consider the difficulty of monitoring the exact dietary intake of PUFAs or of regulating the exact amount ingested in humans. Animal studies offer better control conditions for investigating a possible association between ω-3 PUFAs and aspects of behavior.

It has been shown that a long-term deficiency of ω-3 PUFAs in rats causes changes in dopaminergic and serotonergic neurotransmission. Moreover, major modifications caused by an ω-3 PUFA deficiency were observed in the frontal cortex of animals [34]. These mechanisms are not completely understood. The amount of PUFAs determines the physical qualities of the neuronal membrane, which affects the activity of the associated receptors and transporter molecules [35,36]. In this context, it is important to consider that a depletion of ω-3 PUFAs over at least three generations is required to induce a significant physiological effect on LC-PUFA concentrations (e.g. DHA levels) in the brain of rats [5,9,37].

A reduction of ω-3 PUFAs affects not only the composition of the neuronal membrane, but also the behavior of rodents, including habituation, locomotion and cognition. Several experiments compared the effect of a decreased supply of ω-3 PUFAs on motor activity in rodents. Moriguchi et al. [14] examined the effects of ω-3 PUFA depletion in rats on locomotor activity for five successive days. Normally, locomotor activity slowly decreases with adaptation to the environment (habituation).

Compared to the control group, the ω-3 PUFA-deficient group demonstrated a significant increase in ambulatory time and moving distance on consecutive days. Taken together, these findings suggest that a deficient diet led to a reduced ability to habituate to novel environments, as reflected by an increase in locomotion. This is in line with the findings of Frances et al. [38]. The authors investigated the relationship between a deficiency of LC-PUFAs and habituation in the open field. Their observations indicated that ω-3 PUFA deficient mice were more active than the control group. Moreover, the habituation was significantly decreased between the first and the last day in the control group, but not in the ω-3 PUFA deficient group. These results showed that a reduced intake of ω-3 PUFAs by rodents increased exploration and motor activity [14,38]. Several studies also demonstrated that an ω-3 PUFA deficiency in rodents is associated with impairments in spatial learning [39,40], working memory [41] and olfactory discrimination learning [42] as well as with aggression and depression [43]. Other investigations found that rats fed a diet poor in alpha-linolenic acid (ALA, C18:3 ω-3) demonstrated reduced exploratory activity in new environments, which may be interpreted as a lack of attention to the stimulus environment [44].

In summary, a lack of nutritional essential fatty acids has been suggested to be associated with neurodevelopmental and neuropsychiatric disorders [4–6,45–50]. Significant biological alterations caused by a deficiency of fatty acids lead to this conclusion [1,3,5,6,34,51]. Most human studies investigating the supplementation of LC-PUFAs in the treatment of neuropsychiatric disorders are inconclusive [1,32]. There are limited indications that supplementation of ω-3 PUFAs is neuroprotective, at least in infants [33]. To our knowledge, a few animal experiments concerning LC-PUFAs and cognitive functions have been conducted [39–42]. Two studies have shown a link between ω-3 PUFA deficiency and increased activity [14,38]. However, studies targeting the effects of an ω-3 PUFA deficiency over generations on attention and impulsivity are still lacking.

The aim of the present experiment was to examine the effects of ω-3 PUFAs deficiency on attention functions and impulsive behavior by investigating the eighth generation of ω-3 PUFA depleted Wistar rats. Moreover, we investigated the effects of an ω-3 PUFA adequate diet in these ω-3 PUFA depleted rats on the same behavioral domains. The three-choice-serial-reaction-time-task (3CSRTT) was used for this purpose. This task is based on an established paradigm of attention and impulsivity in rodents, i.e. the five-choice-serial-reaction-time-task (5CSRTT) [52,53].

2. Methods

2.1. Apparatus and testing procedure

2.1.1. 3-choice-serial-reaction-time task (3CSRTT)

The experiment was performed using four ventilated wooden chambers (Campden Instruments, Loughborough, Leicestershire, England) containing a stainless steel chamber (26 cm × 26 cm × 30 cm height) (see Fig. 1). The steel chambers
were lighted by 3-Watt light bulbs. Each chamber was equipped with three holes, which were arranged horizontally in the curved rear wall. The holes were 2 cm above the chamber floor (stainless steel grid); each hole had a diameter of 2 cm and adjacent holes were 6 cm apart. In each hole, an infrared photocell was installed in order to detect a nose poke response of the rat to the hole. In addition, each hole was equipped with a standard light bulb (3 W). The animals were required to respond correctly to a stimulus by a nose poke into one of the three holes. A stimulus was defined as the illumination of a hole by the light bulb, and only one hole at a time could be illuminated. A correct response was rewarded with a food pellet (45 mg dustless sucrose pellets, Bio-Serv, Frenchtown, New Jersey, USA) which was dispensed into a food tray at the front wall (opposite the holes). False responses, premature responses or omissions were punished with a 5-s period of darkness.

The behavioral paradigm consisted of three phases. In the habituation phase, the ambient light was permanently turned on, 10 pellets were baited in the food tray and one pellet was placed in each illuminated hole. The rats were required to habituate to the boxes for 30 min a day. The habituation phase was finished when all pellets were found and collected, which was accomplished within two consecutive days. In the training phase, the rats were required to learn to respond correctly to the stimulus (i.e., random illumination of a hole, once per trial) in order to obtain a food pellet. The stimulus duration was gradually reduced when a rat responded correctly — within one training session of 30 min — in at least 80% of the trials (number of correct trials/total correct and false responses expressed as percent), and the omission rate was less than 20% (number of trials missed/total trials completed expressed as percent). The stimulus duration lasted from 60 s (training level 1) to 1.5 s (final training level). All other parameters were kept constant during the training phase (inter-trial interval ITI of 5 s). In the final (testing) phase, the stimulus duration was 1.5 s and the test sessions were similar to the training sessions except that the ITIs varied randomly between 1.5, 2.5, 3.5, 4.5, 5.5, 6.5, 7.5 and 8.5 s. Each ITI occurred 9 times within one session (72 trials in total). The order in which the groups and rats were tested was randomized in all phases.

2.2. Animals and feeding procedure

Seven generations of Wistar rats were bred in our laboratory and fed an experimental ω-3 PUFA deficient diet. The first generation of female Wistar rats was delivered by Charles River Laboratories (Sulzbach, Germany). The behavioral experiment was carried out with the male offspring of the seventh generation of ω-3 PUFA deficient dams. These dams were randomly assigned to two diet groups: a diet sufficient in ω-3 PUFAs (Sniff, Soest, Germany; based on AIN93G, for details see Table 1) or one deficient in ω-3 PUFAs (see Table 1). The male offspring of dams (not ω-3 PUFA deficient) fed with ω-3 PUFA sufficient diet as described in Table 1 were tested. The specific experimental diets were provided to the dams during prenatal, perinatal and lactation periods, and after weaning to the offspring until the end of the experiment. Therefore, the experimental groups consisted of three distinct groups: 15 ω-3 PUFA deficient male rats of the eighth generation of ω-3 PUFA reduction, prenatally fed with ω-3 PUFA deficient diet, i.e. the ω-3-/ω-3-group; 15 ω-3 PUFA deficient male rats of the eighth generation of ω-3 PUFA reduction, prenatally fed with ω-3 PUFA sufficient diet (ω-3-/ω-3+ group); and finally 15 male rats prenatally fed with ω-3 PUFA sufficient diet (ω-3+/ω-3+ group).

At the beginning of the experiment, male rats of the experimental groups were eight weeks old (body weight approximately 300 g). The rats were housed in standard cages under standard animal laboratory conditions (12:12 h light/dark cycle, room temperature 22 °C, humidity 50%) in the animal laboratories of the University of Regensburg. All treatments, trainings and tests were performed during the light phase between 9 a.m. and 4 p.m. Access to food was restricted since the behavioral paradigm used in this study (3CSRTT) is based on food reinforcement. Water was provided ad libitum. After the training or testing procedures, the rats had free access to food for at least three hours a day. The rats’ weight was carefully controlled, and weight reduction was avoided in order to prevent stress [54,55] and subsequent changes in the dopaminergic system [56]. Rats were monitored daily for health concerns and body weight. After the experiments, rats were sacrificed using carbon dioxide.

Table 1
Fatty acid composition of the experimental diets based on the AIN93G composition (sniff Spezialdiäten GmbH).

<table>
<thead>
<tr>
<th>Product No.</th>
<th>ω-3 sufficient diet customized</th>
<th>ω-3 deficient diet customized</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (Atwater), MJ/kg&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.1</td>
<td>17.1</td>
</tr>
<tr>
<td>kJ% Protein</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>kJ% Carbohydrates</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>kJ% Fat</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>Fatty acids, % of the diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C 6:0</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>C 8:0</td>
<td>0.59</td>
<td>0.62</td>
</tr>
<tr>
<td>C 10:0</td>
<td>0.47</td>
<td>0.49</td>
</tr>
<tr>
<td>C 12:0</td>
<td>3.48</td>
<td>3.64</td>
</tr>
<tr>
<td>C 14:0</td>
<td>1.35</td>
<td>1.41</td>
</tr>
<tr>
<td>C 16:0</td>
<td>0.84</td>
<td>0.85</td>
</tr>
<tr>
<td>C 18:0</td>
<td>0.29</td>
<td>0.28</td>
</tr>
<tr>
<td>C 20:0</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>C 18:1</td>
<td>0.82</td>
<td>0.77</td>
</tr>
<tr>
<td>C 18:2 ω-6</td>
<td>1.54</td>
<td>1.58</td>
</tr>
<tr>
<td>C 18:3 ω-3</td>
<td>0.27</td>
<td>0.01</td>
</tr>
</tbody>
</table>

<sup>a</sup> Physiological fuel value.
2.2. Ethics

All experiments were performed in accordance with the national laws (German law on Protection of Animals) and the principles of laboratory animal care (NIH publication No. 86-23, revised 1996). The rats were handled according to the guidelines of the Federation for European Laboratory Animal Science Associations (FELASA). The rats were monitored daily for health concerns and body weight. Body weight was assessed in order to avoid a reduction of body weight as a consequence of restricted food access. In case of weight loss, the rats were fed individually and were given free access to food for more than 3 hours a day.

2.3. Statistical analysis

2.3.1. Attention parameters

The following parameters were analyzed in order to assess attention processes (see Fig. 2): number of correct responses (i.e. a reaction to an illuminated hole), number of false responses (commission error, i.e. a reaction to a non-illuminated hole), number of omissions (i.e. no reaction to a presented stimulus), percentage of correct responses (i.e. number of correct responses/total correct and false responses expressed as percent) and percentage of omissions (i.e. total number of missed responses/total trials completed expressed as percent).

2.3.2. Impulsivity parameters

With respect to behaviors associated with impulsivity, the following parameters were analyzed: number of premature responses, i.e. nose poke responses during ITI (see Fig. 2), number of panel pushes during intertrial intervals (ITI), i.e. food tray panel pushes during ITI, number of time-out responses, i.e. nose poke responses during time-out period, and total number of perseverative responses.

2.3.3. Activity parameters

Concerning the general activity, the following parameters were analyzed for the comparisons between the diet groups: number of trials completed, number of front beam breaks and number of back beam breaks, the correct response latency, the incorrect response latency and finally the reward collection latency. All findings concerning group differences and comparisons between the diet groups are expressed as means ± standard errors. The statistical analysis of differences between the diet groups was performed using the Mann-Whitney U-Test (between-subject design). An α level of 0.05 was applied. All
statistical analyses were performed using the Statistical Package for Social Sciences 21.0 (SPSS) for Windows.

3. Results

Taken as a whole, we found differences between the ω-3/ω-3- and ω-3+/ω-3+ groups in parameters associated with attentional processes as measured by the 3CSRTT. The rats of the ω-3/ω-3- group made fewer correct responses, more false responses and fewer omissions, although only the number of omissions reached statistical significance (p = .041; Z = −2.046). In addition, the ω-3-/ω-3- group performed worse than the ω-3+/ω-3+ group in regard to the percentage of correct responses and the percentage of omissions. These differences failed statistical significance.

With regard to parameters associated with impulsive behavior, the data show a deterioration in the ω-3-/ω-3- group compared to the ω-3+/ω-3+ group, i.e. the ω-3-/ω-3- group made more premature responses (not significant), more panel pushes during ITI (p = .017; Z = −2.931) and more timeout responses (p < .001; Z = −3.992). In regard to the activity parameters, the correct response latency was significantly longer in the ω-3-/ω-3- group when compared to the ω-3+/ω-3+ group (p = 0.028; Z = −2.198).

The comparison between the ω-3-/ω-3- and the ω-3-/ω-3+ groups showed an improvement in attentional processes in the ω-3-/ω-3+ group as measured by the 3CSRTT. The ω-3-/ω-3+ group made a significantly greater number of correct responses (p = .033; Z = −2.132), showed a significantly higher percentage of correct responses (p = .035; Z = −2.111) and a significantly lower percentage of omissions (p = .033; Z = −2.131).

With regard to the parameters associated with impulsivity, no significant differences in performance could be observed between the ω-3-/ω-3- and ω-3-/ω-3+ groups. This is also true for the activity parameters, with the exception of correct response latency (p = .048; Z = −1.979); all comparisons between the two groups failed statistical significance.

When comparing the ω-3+/ω-3+ and ω-3-/ω-3+ groups, an impairment in parameters measuring impulsive behavior of the ω-3-/ω-3+ group was apparent, since this group made significantly more premature responses (p = .020; Z = −2.331), panel pushes during ITI (p = .001; Z = −3.342) and timeout responses (p < .001; Z = −3.503) compared to the ω-3+/ω-3+ group.

All the other comparisons between groups failed statistical significance. Detailed data (means and standard errors) are provided in Table 2.

4. Discussion

In the present experiment, we used Wistar rats as a transgenerational model of ω-3 PUFA depletion and investigated the effects of this intervention over seven generations on attention functions and impulsive behaviors, as assessed by the 3CSRTT. We also examined behavioral effects of an ω-3 PUFA adequate diet in depleted rats on 3CSRTT performance.

Taken together, the present data show that a transgenerational reduction of dietary ω-3 PUFAs led to substantial changes in attentional processes and behaviors associated with impulsivity compared to rats fed an ω-3 PUFA sufficient diet (ω-3+/ω-3+ group). The impaired performance associated with ω-3 PUFA depletion was partly corrected by the (re)introduction of an ω-3 PUFA sufficient diet beginning in the prenatal period (ω-3+/ω-3+ group), i.e. the ω-3+/ω-3+ group showed an improvement in attentional processes and behaviors associated with impulsivity, as assessed by the 3CSRTT.

Our profile of results is largely in line with previous studies investigating the effects of ω-3 deficiency on cognition. For
example, a dietary deficiency of ω-3 PUFAs was shown to lead to substantial changes in cognitive processes such as spatial learning, working memory and olfactory discrimination learning [39-42].

The present data demonstrated that detrimental effects of transgenerational ω-3 PUFA depletion (ω-3-/ω-3- group) can be at least partly reversed by feeding an ω-3 sufficient diet to dams and their offspring (ω-3-/ω-3+). This finding accords with those of several previous animal studies, which showed a supplementation-associated recovery from behavioral deficits such as impairments in spatial performance, avoidance, anxiety or activity induced by a PUFA deficiency [57-60].

Moreover, the present findings are in accordance with a more recent study by Dervola et al. [61], who investigated the effect of ω-3 PUFA supplementation on the impulsivity and attention in the SHR. In this study, the authors investigated the effects of an ω-3 PUFA enriched diet compared to a control diet on impulsivity, attention and hyperactivity and spontaneous activity in spontaneously hypertensive rats (SHR) and Wistar Kyoto rats (WKY) [61]. Dervola et al. [61] found significant improvements in attention and impulsivity in male SHR fed with the ω-3 PUFA enriched diet compared to those receiving a control diet. Although the authors used SHR rather than Wistar rats, these findings are essentially in line with the present results. Dervola et al. [61] analyzed the levels of dopamine, serotonin and their metabolites in the neostriatum using high performance liquid chromatography. In male SHR, a significant increase in dopamine and serotonin turnover, as expressed by increased rates of monoamine metabolites relative to neurotransmitters, was found compared to rats fed the control diet. These changes in neurotransmitter signaling were correlated with dietary effects at the behavioral level [61].

The present findings, together with the study of Dervola et al. [61], support the hypothesis that an ω-3 PUFA deficiency affects dopaminergic and serotonergic neurotransmission [35,36,62-64] since it is known that these neurotransmitters are involved in the modulation of attention and impulse control [34,51,65,66]. Interestingly, in the present study these cognitive domains were demonstrated to be influenced by the variation in quantity of ω-3 PUFAs, specifically by an ω-3 PUFA deficiency over generations and the treatment with an ω-3 PUFA sufficient diet in ω-3 deficient rats.

Both the present and previous findings regarding behavioral and neurobiological effects of dietary modifications of ω-3 PUFAs could be taken to imply a direct effect of dietary ω-3 PUFA intake on neurobiological systems (e.g. alterations in monoaminergic neurotransmitter systems) and cognitive functioning. However, there are alternative explanations. For example, the ω-3 PUFA deprivation in rats over generations may cause other developmental changes or induce an unnaturally high inflammatory level, which may have a direct impact on cognitive capacity [67]. This raises the question of whether an ω-3 PUFA deficient animal model produced over several generations is suitable for studies investigating the influence of LC-PUFA on behavior. The biosynthesis of DHA from ALA in rodents is more effective than in nonhuman primates [68], i.e. a longer period of ω-3 PUFA deprivation (more than one generation) is needed to achieve a reduced DHA concentration in the brain [37,67]. A recent study investigated the effects of ω-3 PUFA reduction over four generations in Wistar rats and failed to find any significant changes in attention and impulsivity [69].

Since the present study showed significant effects on attention and impulsivity parameters in ω-3 PUFA-deficient rats, when compared to ω-3-nondeficient rats, and improved functioning after prenatal administration of an ω-3 sufficient diet, it would be interesting to perform further investigations using higher doses of ω-3 PUFAs.

The present data show no marked changes in motor activity, either by experimental reduction and/or supplementation of ω-3 PUFA availability. Only two of many statistical comparisons reached statistical significance. This indicates that the effects of ω-3 PUFA reduction and/or supplementation on attentional processes and impulsive behavior are not caused by changes in activity [70]. This finding of partial independence of attentional performance, impulsivity-related and activity-related parameters in the 3CSRTT is not new and may be taken to demonstrate the partial independence of underlying neuro-modulatory mechanisms in the brain [53,71]. Unaltered motor activity together with reductions in impulsive behavior may reflect improvements in decision making processes in the ω-3 PUFA sufficient rats compared to the ω-3 PUFA deficient groups [70]. Our findings are not in agreement with Enslen et al. [44], who reported a reduction in motor activity following a depletion of ω-3 PUFAs. A possible explanation for these discrepant findings may be an influence of ω-3 PUFAs on the motivational state [72,73]. To elucidate this issue, these results should be replicated in future experiments that control for motivational aspects, for example by using a constant number of trials instead of time as termination criterion in the 3CSRTT.

In conclusion, the present findings demonstrated that transgenerational dietary changes in regard to ω-3 PUFAs led to changes in attentional processes and impulsive behavior, supporting the hypothesis that ω-3 PUFAs play a role in cognitive and behavioral processes. It is important to note that these behavioral changes were observed following a transgenerational reduction of ω-3 PUFAs over more than seven generations. This model cannot easily be translated to humans, since such dramatic reductions in ω-3 PUFA intake are unlikely to occur in humans. The present data regarding cognitive and behavioral effects of ω-3 PUFA manipulations in rats do not therefore allow any conclusions for humans. Nutritional recommendations pertaining to the use of ω-3 PUFA supplementation as a supportive therapy in neuropsychiatric disorders cannot be based on this kind of evidence. However, the present findings suggest the potential usefulness of a transgenerational animal model of ω-3 PUFA deficiency in regard to various cognitive and behavioral domains such as attention, impulse control, spatial recognition, discrimination, reward preference, memory and learning.

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References


