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# Persistent Aggressive Behaviour From Childhood to Adolescence: The Influence of Environmental Tobacco Exposure and the Protective Role of Fish Consumption

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## ABSTRACT

**Background:** Understanding changes in aggressive behaviour throughout child development is crucial for identifying effective intervention strategies. This study investigates children's aggressive behaviour in a longitudinal cohort and explores the role of environmental tobacco exposure and fish consumption as potential risk and protective factors, respectively, for persistent aggression in children.

**Methods:** This study involved 452 children from the Chinese Jintan Cohort. Aggressive behaviour was assessed at ages 6 and 12 years using the child behaviour checklist (CBCL) and the Reactive–Proactive Aggression Questionnaire (RPQ), respectively. Information on lifestyle habits and living environment, including parental smoking, was collected via questionnaires. Linear regression was employed to investigate the association between childhood and adolescence aggressive behaviour with relevant covariates adjusted. Subsequently, we conducted interaction analyses to explore the moderating effects of parent smoking and fish consumption on the association.

**Results:** We identified no significant association between childhood and adolescent aggression in the entire sample. Interaction analysis revealed environmental tobacco exposure as a moderator for the association. Specifically, persistent reactive and total aggression across development was only observed among those with environmental tobacco exposure (reactive: $\beta = 0.549$, $p = 0.020$; total: $\beta = 0.654$, $p = 0.035$). Furthermore, within the parent smoking subgroup, freshwater fish consumption at the age of 12 showed a marginally significant interaction with childhood aggression (reactive: $p = 0.061$; total: $p = 0.095$). A significant longitudinal association for aggression was found only among those consuming fish less frequently at the age of 12 years (reactive: $\beta = 0.927$, $p = 0.002$; total: $\beta = 1.082$, $p = 0.006$).

**Conclusion:** Our findings suggest exposure to environmental tobacco as a contributing factor to the lasting presence of aggressive behaviour during children's development, whereas freshwater fish consumption shows potential protective effects.

## 1 | Introduction

Childhood is a period of development where children undergo profound physical, cognitive and emotional changes (Likhar, Baghel, and Patil 2022). From preschool age to early adolescence, many children who exhibit temperamental problems such as irritability and impulsivity often mature into well-adjusted individuals (Zarrella et al. 2017). However, this developmental trajectory is not universal (Moffitt 2017; Nagin, Farrington, and Moffitt 1995). A subset of children continues to demonstrate aggressive and/or antisocial behaviours throughout later development (Hyde, Shaw, and Hariri 2013; Rudolph et al. 2014), defined as persistent aggressive behaviour. Understanding what accounts for the continuity of aggressive and antisocial behaviour throughout childhood, and why some children maintain these behaviours into adolescence while others do not, is essential for developing effective intervention strategies appropriate to the developmental period (Hendriks et al. 2018; Wong, Francesconi, and Flouri 2021).

Extensive research has identified exposure to tobacco, including prenatal and early-life exposure, as a significant risk factor for the development of behavioural problems in children (Glenn, Ragno, and Liu 2023; H. S. Yang et al. 2018). Maternal smoking during pregnancy has been associated with a range of adverse outcomes, including increased risk of conduct disorders and delinquent behaviours in children (Farrington, Gaffney, and Ttofi 2017; Ruisch et al. 2018). Postnatal exposure to second-hand smoke has also been linked with increased conduct problems and aggression. In a longitudinal cohort of 1035 children, increases in household tobacco exposure were prospectively associated with increases in conduct problems, proactive and reactive aggression as well as school indiscipline and drop-out risks (L. Pagani et al. 2017). Additionally, children who were ever exposed at any given time or always exposed to second-hand smoking were found to have higher odds of having attention-deficit/hyperactivity disorder (ADHD) symptoms (Huang et al. 2021; Lin et al. 2021). These findings suggest that exposure to environmental tobacco can have long-lasting effects on children's behavioural development, underscoring the importance of addressing tobacco exposure as a modifiable risk factor in efforts to prevent and mitigate antisocial behaviour.

Lifestyle factors, such as exercise (Ouyang and Liu 2023), sleep (J. Liu et al. 2022) and diet (Choy 2023), have been shown to be associated with aggressive behaviour. There is an increasing interest in the use of nutritional supplements to reduce aggressive behaviour in children. One recent meta-analysis (Raine and Brodrick 2024) specifically examined the role of omega-3 supplementation in reducing aggression. Analyses from 29 RCTs encompassing 3918 individuals revealed a significant effect size of 0.22. There was no evidence of publication bias and sensitivity analyses confirmed the findings. Although there was significant heterogeneity, moderator analyses were largely nonsignificant, indicating that beneficial effects are obtained across age, gender, recruitment sample, baseline diagnoses, treatment duration and dosage. Effects also applied to reactive as well as proactive forms of aggression. Overall, results from these 35 independent RCT samples provide substantial evidence that omega-3 supplementation can lead to modest short-term reductions in aggression. This raises the question of whether fish consumption can protect against the continuing development of aggressive behaviour in children who are exposed to other health risk factors for aggression, including nicotine exposure.

The relation between fish consumption and aggression may be particularly relevant to those exposed to smoking. Smokers have reduced levels of DHA and EPA (Scaglia et al. 2016), two key constituents of omega-3. There is some evidence that this relationship is causal because a randomized controlled trial (RCT) providing omega-3 supplements for 1 month led to both a significant reduction in tobacco craving and most notably a significant reduction in daily smoking (Rabinovitz 2014), a finding replicated in another RCT involving heavy smokers (Sadeghi-Ardekani, Haghighi, and Zarrin 2018). Smoking may lower omega-3 levels because omega-3 fatty acids are susceptible to oxidation, and it is known that smoking increases the peroxidation of omega-3 fatty acids (Scaglia et al. 2016). Given this background, it can be hypothesised that exposure to cigarette smoking in adolescents could lower their omega-3 levels and predispose to aggression, particularly in those with smoking exposure, but that fish consumption in this group could protect against aggression, given that fish is a major source of dietary omega-3.

The current study has the following aims to better understand the development of aggressive behaviour in children. First, we aim to examine the extent of continuity of aggressive behaviour from ages 6 to 12, investigating the stability and change of such behaviour over time. Second, we aim to evaluate the extent to which environmental tobacco exposure, as a risk factor, influences this developmental continuity, particularly with respect to reactive and proactive aggression that constitute two main forms of aggressive behaviour (Raine et al. 2006). Third, we aim to examine whether fish consumption, as a source of omega-3 fatty acids, can reduce the continuity of aggression in children exposed to environmental tobacco. By addressing these aims, we hope to provide insights into the future development of targeted intervention strategies that can effectively reduce aggressive behaviour among at-risk children (Farrington 2007).

## 2 | Methods

### 2.1 | Study Design

This study employed data from the longitudinal China Jintan Child Cohort Study. The aim was to investigate the interactions between lifestyle, social and environmental factors and children's neurobehavioral outcomes. During the period 2004–2005, they recruited 1656 preschool children aged three to five years from Jintan City, Jiangsu province, China, who were representative of the full geographical, social and economic profiles of the area. The first wave of assessments was conducted during 2005–2007 when the children were approximately 6 years old. The second wave of data collection (Wave II) occurred during 2011–2013 when participants were aged approximately 12 years, and we successfully followed 1110 of the previous participants. At both waves, data were collected from children, parents and teachers through questionnaires and psychophysiology tests. Detailed information on the Jintan Study, including subjects, recruitment and procedures, can be accessed elsewhere (Liu et al. 2010, 2012, 2015).

## 2.2 | Study Participants and Procedures

The current study included 452 participants who had complete data on demographic information, aggression across two waves and lifestyle factors of interest such as exposure to environmental tobacco and fish consumption. All childhood variables were reported by the parents and were collected at approximately 6 years of age. Specifically, with children at different grades during enrolment, the questionnaire was administered across three years, from 2005 to 2007, when the children graduated from preschool. Adolescence information was collected from children's self-report at approximately 12 years of age, when they were in the last semester of their sixth grade. Similarly, because of grade differences, the data collection spanned from 2011 to 2013. Detailed demographic characteristics are summarised in Table 1. Written informed consent was obtained from the parents at both collection waves, whereas verbal informed assent was obtained from the adolescents prior to the second wave of collection. Institutional review board approval was obtained from the University of Pennsylvania and the ethical committee for research at Jintan Hospital in China.

## 2.3 | Measures

### 2.3.1 | Childhood Aggression at Age 6

We used the aggressive behaviour subscale from the children's behaviour checklist (CBCL) for ages 1.5–5 (Achenbach and Edelbrock 1991) as a measurement for childhood aggression. CBCL is part of the Achenbach System of Empirically Based Assessment (ASEBA) (Rescorla 2005) and is one of the most widely used (Rescorla et al. 2012) and validated (Achenbach and Rescorla 2000) measures for assessing children's behavioural and emotional functioning. The aggressive behaviour subscale was calculated from 19 items from CBCL, each rated on a 3-point scale (0, not true; 1, sometimes true; and 2, often true). The subscale sums to 38 points in total, with a higher score indicating more severe aggressive behaviour. The Chinese version of the CBCL was used in this study. It has been well validated (Leung et al. 2006; J. Liu et al. 2011) and has been used in various studies (Liu et al. 2001, 2014).

### 2.3.2 | Adolescence Aggression at Age 12

Adolescence aggression was measured using the Chinese version of the Reactive and Proactive Aggression Questionnaire (RPQ) (Tuvblad et al. 2016). The instrument contains 23 items that were designed to measure reactive (12 items) and proactive (11 items) aggressions (Raine et al. 2006). Proactive aggression is generally characterised as instrumental, organised and 'cold-blooded', whereas reactive aggression is mostly characterised by impulsive, emotional and 'hot-blooded' aggression (Raine et al. 2006). Each item has a three-point response format (0, never; 1, sometimes; and 2, often). The scores were summed to derive proactive, reactive and total aggression scores, totalling 22, 24 and 46 points, respectively. Higher aggression scores indicate more severe aggressive behaviour. The Chinese version of the RPQ has been validated (Tuvblad et al. 2016) with a Cronbach's alpha of 0.810–0.893 for the three aggression scores.

### 2.3.3 | Environmental Tobacco Exposure Via Parental Smoking

The environmental tobacco exposure was assessed via the smoking status of parents, which was collected as part of the general information questionnaire at Wave II around age 12. Tobacco exposure was coded as 'Yes' when at least one of the child's parents smokes.

### 2.3.4 | Fish Consumption

The childhood fish consumption frequency was rated among 'often', 'sometimes', 'occasionally' and 'seldom'. In the current study, we recoded 'often' and 'sometimes' as 'Often' and the remaining two as 'Less Often'. Adolescence fish consumption was split into specific fish types including freshwater fish, seawater fish and shellfish. The children would select among frequencies 'more than once a day', '4–7 times per week', '1–3 times per week', 'every 2 weeks' and 'never or rarely'. The first three options were recoded as 'Often', whereas the remaining two were recoded as 'Less Often'.

## 2.4 | Covariates

Five sociodemographic variables, including gender, residence location (suburb, city and rural), children's grade, children's age at childhood data collection and parent's average monthly income, were included as covariates for the initial regression analysis. Children's age at childhood were calculated by the time lapse between June 1st of the data collection year and their date of birth, with the data collection year (2005–2007) determined by the children's grade. Parents' average income was collected at Wave II and was initially rated among options, '< 500', '500–1000', '1000–2000', '2000–3000', '3000–5000', '5000–10000', '> 10,000', all in Chinese RMB. The incomes of parents were reported separately and were averaged in this study to derive averaged parent income. Violence exposure at childhood and adolescence were further included as covariates in the exploration of potential risk factors for the persistence of longitudinal aggression. Violence at both waves were measured through the conflict tactics scales (CTS) (Straus and Hamby 1997; Straus et al. 1996). Fish consumption at childhood was further controlled for in fish-relevant analyses. Throughout analysis, the reactive aggression score was added as a covariate in the analysis of proactive aggression and vice versa.

## 2.5 | Statistical Analyses

Means, medians and standard deviations (SD) were used to describe the participants' demographic characteristics wherever appropriate. The differences between smoking-exposed and nonsmoking subgroups as well as between different fish consumption groups were compared using *t*-tests and chi-square tests (Table 1, Supp. Table S1). Aggression at both waves were

**TABLE 1** | Characteristics of participants.

| | Total ($N = 452$) | Parent nonsmoking ($N = 171$) | Parent smoking ($N = 281$) | $t/\chi^2$ | $p$ |
|---|---|---|---|---|---|
| Age at childhood, mean (SD) (Range) | 6.02 (0.42) (4.63–8.32) | 6.04 (0.44) (4.63–7.85) | 6.00 (0.41) (5.21–8.32) | −0.908 | 0.364 |
| Gender, *N* (%) | | | | 0.001 | 0.973 |
| Male | 241 (53.3%) | 91 (53.2%) | 150 (53.4%) | | |
| Female | 211 (46.7%) | 80 (46.8%) | 131 (46.6%) | | |
| Residence location, *N* (%) | | | | 3.502 | 0.174 |
| Rural | 69 (15.3%) | 22 (12.9%) | 47 (16.7%) | | |
| Suburban | 177 (39.2%) | 76 (44.4%) | 101 (35.9%) | | |
| City | 206 (45.6%) | 73 (42.7%) | 133 (47.3%) | | |
| Grade, *N* (%) | | | | 0.446 | 0.800 |
| 4th | 192 (42.5%) | 76 (44.4%) | 116 (41.3%) | | |
| 5th | 127 (28.1%) | 46 (26.9%) | 81 (28.8%) | | |
| 6th | 133 (29.4%) | 49 (28.7%) | 84 (29.9%) | | |
| Average parent monthly income, *N* (%) | | | | 1.269 | 0.260 |
| ≤ 3000 RMB (equivalent to 400 USD) | 212 (46.9%) | 86 (50.3%) | 126 (44.8%) | | |
| > 3000 RMB | 240 (53.1%) | 85 (49.7%) | 155 (55.2%) | | |
| Fish consumption at childhood, *N* (%) | | | | 0.381 | 0.537 |
| Less often | 68 (15.0%) | 28 (16.4%) | 40 (14.2%) | | |
| Often | 384 (85.0%) | 143 (83.6%) | 241 (85.8%) | | |
| Freshwater fish consumption at adolescence, *N* (%) | | | | 0.003 | 0.956 |
| Less often | 216 (47.8%) | 82 (48.0%) | 134 (47.7%) | | |
| Often | 236 (52.2%) | 89 (52.0%) | 147 (52.3%) | | |
| Seawater fish consumption at adolescence, *N* (%) | | | | 0.366 | 0.545 |
| Less often | 309 (68.4%) | 114 (66.7%) | 195 (69.4%) | | |
| Often | 143 (31.6%) | 57 (33.3%) | 86 (30.6%) | | |
| Aggressive behaviour at childhood, mean (SD) (range) | 8.39 (5.78) (0–30) | 8.56 (5.95) (20–23) | 8.29 (5.68) (0–30) | 0.392 | 0.695 |
| Proactive aggression at adolescence, mean (SD) (range) | 1.23 (2.70) (0–15) | 1.19 (2.54) (0–14) | 1.26 (2.80) (0–15) | 0.088 | 0.930 |
| Reactive aggression at adolescence, mean (SD) (range) | 5.00 (3.85) (0–22) | 5.04 (3.96) (0–19) | 4.98 (3.79) (0–22) | −0.015 | 0.988 |
| Total aggression at adolescence, mean (SD) (range) | 6.24 (5.71) (0–31) | 6.23 (5.61) (0–29) | 6.24 (5.78) (0–31) | 0.028 | 0.978 |

*Note:* Childhood variables measured at age 6, adolescence variables measured at age 12.
Abbreviation: *SD*, standard deviation.

log-transformed to improve normality. Violent behaviour were square-root transformed following the previous literature (Portnoy et al. 2018). Normality of the data and potential outliers were examined through visual inspection of the Q–Q plot. Linear regression was used to examine the association between childhood and adolescence aggression in the whole sample and under subgroups. Interaction analyses were conducted using the PROCESS MACRO software Model 1, with tobacco exposure or fish consumption as moderators. The variables were not centred in the moderation analyses. A *p*-value less than 0.05 was considered significant, whereas a *p*-value larger than 0.05 but smaller than 0.10 was regarded as marginally significant. All analysis were performed using SPSS, version 26 (IBM).

## 3 | Results

### 3.1 | Descriptive Statistics

We included 452 children in this study, of whom 211 (46.7%) were female. The children were on average 6.02 (SD = 0.42)

years old at the time of childhood measurement during 2005–2007. The average parent-report aggression during childhood was 8.39 (SD = 5.78), whereas that of the self-report proactive, reactive and total aggression at adolescence were 1.23 (SD = 2.70), 5.00 (SD = 3.85) and 6.24 (SD = 5.71), respectively. Among the participants, 281 (62.2%) children had at least one of their parents smoking. Around half of the children eat freshwater fish relatively often, for at least once per week. Detailed characteristics, family information and lifestyle habits of the children are described in Table 1. We observed no differences between children with or without environmental tobacco exposure in all demographic variables.

### 3.2 | Association of Aggression Across Age

We first investigated whether aggressive behaviour in childhood persists as the children step into adolescence. We used childhood aggression as a predictor for adolescence proactive, reactive and total aggression, with age, gender, living location, parent's income and grade controlled as covariates. Proactive aggression was additionally controlled in the analysis of reactive aggression and vice versa. We observed no significant associations between childhood and adolescence aggression, except for a marginally significant association for reactive aggression ($\beta = 0.294$, $p = 0.067$, Table 2).

### 3.3 | Interaction Effects of Environmental Tobacco Exposure Via Parent Smoking

We further explored factors that contribute to the association. Environmental tobacco exposure showed a significant interaction with childhood aggression in the model of reactive and total aggression (reactive: $\beta = 0.637$, $p = 0.039$; total: $\beta = 0.785$, $p = 0.049$), with violence exposure at both waves additionally controlled (Figure 1). Stratified analysis in parent smoking and nonsmoking subgroups further revealed that childhood aggression significantly predicts adolescence aggression only in children exposed to tobacco at the age of 12 years (reactive: $\beta = 0.549$, $p = 0.020$; total: $\beta = 0.654$, $p = 0.035$, Table 2).

### 3.4 | Moderation Effects of Fish Consumption

We then focused on children exposed to environmental tobacco (parent smoking) ($N = 281$) and explored potential lifestyle habits that may potentially mitigate the persistence of aggression along development. Among the various factors that we examined (including breakfast, exercise, sleep and diet), freshwater fish consumption at adolescence exhibits a marginally significant interaction with childhood aggression (reactive: $\beta = -0.722$, $p = 0.061$; total: $\beta = -0.843$, $p = 0.095$, Figure 2, Table 3). Specifically, children who consumed freshwater fish

**TABLE 2** | Associations between childhood and adolescence aggression and the interaction with exposure to tobacco.

| | Proactive aggression | | Reactive aggression | | Total aggression | |
|---|---|---|---|---|---|---|
| **Adolescent** | **β (SE)** | ***p*** | **β (SE)** | ***p*** | **β (SE)** | ***p*** |
| Total effects | | | | | | |
| Childhood aggression[a] | −0.098 (0.151) | 0.514 | 0.294 (0.160) | 0.067 | 0.297 (0.211) | 0.160 |
| Interaction analysis | | | | | | |
| Intercept | 0.575 (0.472) | 0.223 | −0.180 (0.488) | 0.713 | 0.446 (0.629) | 0.478 |
| Age | −0.030 (0.031) | 0.335 | 0.014 (0.032) | 0.665 | −0.013 (0.041) | 0.758 |
| Gender | −0.129 (0.025) | < 0.001 | 0.079 (0.026) | 0.003 | −0.019 (0.033) | 0.576 |
| Residence | −0.007 (0.017) | 0.671 | 0.018 (0.018) | 0.315 | 0.021 (0.023) | 0.372 |
| Grade | −0.058 (0.016) | < 0.001 | 0.083 (0.017) | < 0.001 | 0.063 (0.021) | 0.003 |
| Parents' income | −0.020 (0.011) | 0.067 | 0.013 (0.012) | 0.253 | −0.002 (0.015) | 0.919 |
| Childhood parents' violence | −0.004 (0.008) | 0.675 | −0.019 (0.009) | 0.030 | −0.032 (0.011) | 0.004 |
| Adolescence parents' violence | 0.006 (0.006) | 0.326 | 0.032 (0.007) | < 0.001 | 0.051 (0.008) | < 0.001 |
| Childhood aggression | 0.023 (0.234) | 0.923 | −0.018 (0.241) | 0.941 | −0.053 (0.312) | 0.864 |
| Exposure to tobacco | 0.304 (0.510) | 0.551 | −1.091 (0.525) | 0.038 | −1.350 (0.677) | 0.047 |
| Interaction[b] | −0.180 (0.300) | 0.549 | 0.637 (0.308) | 0.039 | 0.785 (0.397) | 0.049 |
| Conditional effects | | | | | | |
| Exposure to tobacco[c] | | | | | | |
| Yes | −0.157 (0.213) | 0.461 | 0.619 (0.218) | 0.005 | 0.732 (0.281) | 0.010 |
| No | 0.023 (0.234) | 0.923 | −0.018 (0.241) | 0.941 | −0.053 (0.312) | 0.864 |

*Note:* Linear regression models were employed. Childhood variables measured at age 6 and adolescence variables measured at age 12.
Abbreviations: β, Unstandardised Coefficients; SE, Standardized Error.
[a]Controlled for age, gender, residence location, grade and parents' income.
[b]Interaction between childhood aggression and exposure to tobacco, additionally controlled for parents' violence at both childhood and adolescence.
[c]Conditional coefficients.

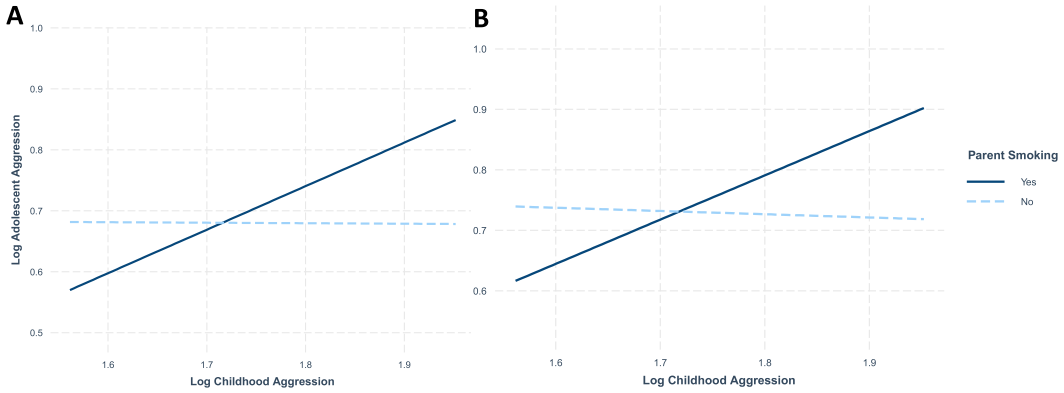


**FIGURE 1** | Interaction plot of childhood aggression with parent smoking (A) on reactive aggression and (B) on total aggression. Linear regression models were employed with age, gender, residence location, grade, parents' income and parents' violence at both childhood and adolescence controlled. [Colour figure can be viewed at wileyonlinelibrary.com]

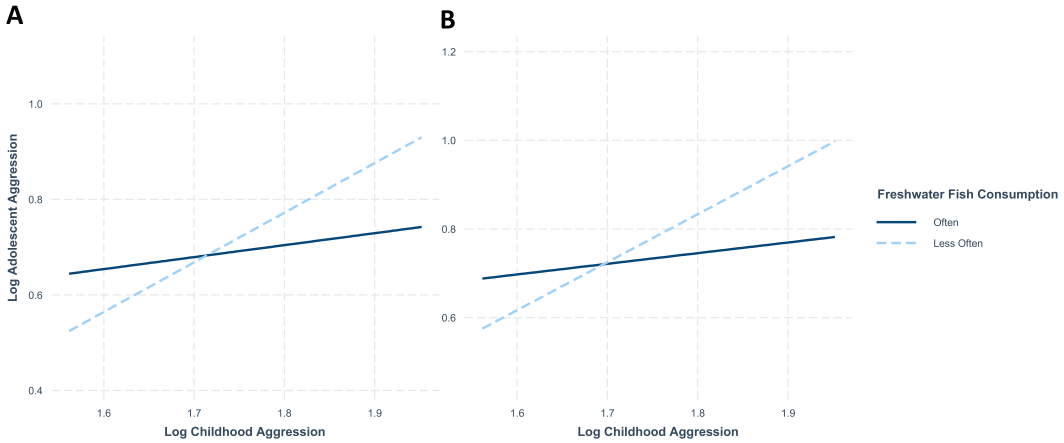


**FIGURE 2** | Interaction plot of childhood aggression with freshwater fish consumption (A) on reactive aggression and (B) on total aggression. Linear regression models were employed with age, gender, residence location, grade, parents' income, parents' violence at both childhood and adolescence and fish consumption frequency at childhood controlled. [Colour figure can be viewed at wileyonlinelibrary.com]

less often (less than once per week) exhibited persistent aggression from childhood to adolescence (reactive: $\beta = 0.9273$, $p = 0.002$; total: $\beta = 1.082$, $p = 0.006$), whereas those who consumed freshwater fish more often showed no such association (Table 3). No interaction effect was identified for seawater fish. We did not examine the effects of shellfish, given that all participants reported rare shellfish consumption. We observed no differences in demographic variables between the fish consumption subgroups (Supp. Table S1).

## 4 | Discussion

This study utilised data from the longitudinal China Jintan Child Cohort Study to examine the impact of environmental tobacco exposure and fish consumption on aggressive behaviour, following 452 children from preschool age (3–5 years) to early adolescence (around age 12). Although childhood aggression did not predict adolescent aggression in the overall sample, we found that persistent reactive aggression and total aggression across development existed only among children with environmental tobacco exposure. Additionally, children who consumed freshwater fish less often exhibited a marginally significant longitudinal association in aggression, whereas those who consumed more fish did not, indicating that frequent freshwater fish consumption may have mitigated the effects of tobacco exposure. These findings underscore the importance of addressing modifiable lifestyle factors to prevent persistent antisocial behaviours in children.

The lack of persistence in aggression from childhood to adolescence in the overall sample may be attributed to significant developmental changes during this period. Research indicates that early childhood is marked by rapid developmental changes that can influence behaviour trajectories (C. Chen 2024). Additionally, in our study, the preschool aggression measure, CBCL, was based on parent reports, whereas the age 12 aggression outcome was based on self-reported RPQ. It is well-documented that parent and child reports of behaviour often do not correlate well even when assessed at the same point in time (Caqueo-Urízar et al. 2022; Y. Y. Chen et al. 2017). For example, in a cross-sectional study with 8154 children, significant differences were found in the way parents and children perceived and reported children's emotional–behavioural difficulties (Van Roy et al. 2010). Therefore, it could be possible that cross-informant

**TABLE 3** | Moderation effects of freshwater fish consumption on associations between childhood and adolescence aggression in children with exposure to tobacco.

| | Proactive aggression | | Reactive aggression | | Total aggression | |
|---|---|---|---|---|---|---|
| **Adolescent** | **β (SE)** | ***p*** | **β (SE)** | ***p*** | **β (SE)** | ***p*** |
| Total effects | | | | | | |
| Childhood aggression[a] | −0.153 (0.231) | 0.510 | 0.549 (0.235) | 0.020 | 0.654 (0.308) | 0.035 |
| Interaction analysis | | | | | | |
| Intercept | 1.222 (0.632) | 0.054 | −1.726 (0.638) | 0.007 | −1.294 (0.837) | 0.123 |
| Age | −0.043 (0.041) | 0.294 | 0.019 (0.041) | 0.651 | −0.016 (0.054) | 0.764 |
| Gender | −0.057 (0.020) | 0.006 | 0.104 (0.036) | 0.002 | −0.000 (0.043) | 0.994 |
| Residence | −0.007 (0.022) | 0.739 | 0.012 (0.022) | 0.582 | 0.012 (0.029) | 0.681 |
| Grade | −0.057 (0.020) | 0.006 | 0.066 (0.021) | 0.002 | 0.039 (0.027) | 0.152 |
| Parents' income | −0.030 (0.014) | 0.034 | 0.025 (0.009) | 0.082 | 0.005 (0.019) | 0.778 |
| Childhood parents' violence | −0.004 (0.11) | 0.696 | −0.017 (0.011) | 0.136 | −0.031 (0.015) | 0.035 |
| Adolescence parents' violence | 0.011 (0.009) | 0.191 | 0.028 (0.009) | 0.001 | 0.053 (0.011) | < 0.001 |
| Childhood fish consumption | 0.031 (0.046) | 0.507 | | | −0.098 (0.061) | 0.109 |
| Sea fish consumption | 0.018 (0.037) | 0.621 | | | −0.020 (0.049) | 0.683 |
| Childhood aggression | −0.307 (0.296) | 0.300 | 0.927 (0.296) | 0.002 | 1.082 (0.387) | 0.006 |
| Freshwater fish consumption | −0.495 (0.647) | 0.445 | 1.261 (0.653) | 0.055 | 1.437 (0.858) | 0.095 |
| Interaction[b] | 0.269 (0.3780) | 0.479 | −0.722 (0.384) | 0.061 | −0.842 (0.503) | 0.095 |
| Conditional effects | | | | | | |
| Freshwater fish consumption[c] | | | | | | |
| Less often | −0.307 (0.296) | 0.300 | 0.927 (0.296) | 0.002 | 1.082 (0.387) | 0.006 |
| Often | −0.038 (0.306) | 0.901 | 0.205 (0.311) | 0.510 | 0.240 (0.407) | 0.556 |

*Note:* Linear regression models were employed. Childhood variables measured at age 6 and adolescence variables measured at age 12.
Abbreviations: β, Unstandardised Coefficients; SE, Standardized Error.
[a]Controlled for age, gender, residence location, grade, parents' income, and parents' violence at both childhood and adolescence.
[b]Interaction between childhood aggression and freshwater fish consumption, additionally controlled for fish consumption frequency at childhood and sea fish consumption.
[c]Conditional coefficients.

agreement was not high when the two measurements, CBCL and RPQ, were administered across an 8-year span. This discrepancy in reporting sources could contribute to the observed lack of longitudinal continuity in aggressive behaviour. Such differences highlight the importance of considering multiple informants and the developmental context when interpreting behavioural data and formulating intervention strategies (De Los Reyes 2013; De Los Reyes et al. 2013).

Despite the overall lack of longitudinal continuity in aggression, we identified a significant interaction between environmental tobacco exposure and childhood aggression, suggesting that tobacco exposure may have contributed to some persistence of aggressive behaviour. The interaction effect was more evident for reactive than proactive aggression, which may be due to low scores on proactive aggression inducing a floor effect which precluded significant results for this latter form of aggression. The finding for reactive aggression aligns with previous research highlighting tobacco exposure as a consistent risk factor for aggression across different developmental stages (Glenn, Ragno, and Liu 2023; Rückinger et al. 2010). A longitudinal study with 2055 children demonstrated that compared to children never exposed to tobacco, children exposed to continuous or transient levels of postnatal second-hand smoke from the age of 17 to 86 months scored higher on self-reported aggressive behaviour and teacher-rated antisocial behaviour in the fourth grade (L. S. Pagani and Fitzpatrick 2013). Similarly, another prospective longitudinal cohort with seven waves of data found that parental smoking independently predicted higher levels of child-externalising behaviours even after controlling for demographic correlates of smoking (Steeger et al. 2019). In addition, children exposed to tobacco in early life were found to be at greater risk of exhibiting high-risk behaviours later on (Gonzalez-Sicilia, Derevensky, and Pagani 2020). These longitudinal studies suggest a potential causal link between environmental tobacco exposure and aggression. Tobacco smoke is composed of thousands of chemical compounds, many of which are neurotoxicants (J. Liu 2011; J. Liu et al. 2013). Nicotine, in particular, is a neuroteratogen that can contribute to the death of brain cells or structural changes in specific brain regions during brain development, potentially resulting in neurobehavioral problems in offspring (Castro, Lotfipour, and Leslie 2023). Although we only measured parental smoking behaviour once, it is safe to assume that, given the addictive nature of smoking

(Roh 2018), parents who smoked when their children were young were likely to continue smoking as their children grew older, potentially maintaining the association between environmental tobacco exposure and aggression over time.

Previous research has suggested that dietary factors, such as omega-3 fatty acids found in fish, might play a role in mitigating aggressive behaviour in children (Choy and Raine 2018; Gajos and Beaver 2016; Raine et al. 2015). In our study, we observed a trend where frequent consumption of freshwater fish during adolescence appeared to attenuate the association between early childhood aggression and later aggressive behaviour among children with tobacco exposure. No such effect was observed for seawater fish consumption. This may be due to the fact that consumption of freshwater fish was higher than seawater fish consumption (see Table 1) because of the geographic location of Jintan City. This finding for freshwater fish consumption aligns with the hypothesis that omega-3 fatty acids may have a protective effect on behavioural outcomes.

Why would omega-3 supplementation be expected to reduce aggressive behaviour? At one level, it is well-known that there is a significant neurobiological basis to aggressive and violent behaviour (Blair 2022; Williams et al. 2018; Y. Yang and Raine 2009). At a mechanistic level, it is also known that omega-3 is a long-chain fatty acid that plays a critical role in brain structure and function. It plays multiple roles, making up approximately 35% of the cell membrane, enhancing neurite outgrowth, regulating both neurotransmitter functioning and gene expression and being involved in neurogenesis and nerve cell signalling (Bazinet and Laye 2014; Diaz, Mesa-Herrera, and Marin 2021; McNamara and Carlson 2006). Omega-3 also reduces inflammatory processes in the brain and plays a significant role in cerebral blood flow (McNamara et al. 2018; von Schacky 2021). Structural and functional brain imaging studies on humans have further documented that omega-3 can upregulate a variety of brain regions, with no evidence for any detrimental effect (McNamara and Almeida 2019). As such, given the undeniable fact that omega-3 is pervasively involved in multiple facets of neuronal biology, it is reasonable to believe that omega-3 supplementation could play a causal role in reducing aggression by upregulating brain mechanisms that may be dysfunctional in such individuals, given the assumption that there is, in part, a neurobiological basis to aggression. However, further research is needed to establish causal relationships and determine optimal dietary recommendations for reducing aggression in at-risk populations.

One important strength of our study is that we employed a longitudinal design, allowing for the examination of behavioural trajectories over time, which enhances the robustness of our findings (Farrington 2015). However, several limitations should be noted. Although we observed the potential protective role of frequent freshwater fish consumption during adolescence in mitigating the persistence of aggressive behaviour among children with exposure to environmental tobacco, this was a marginally significant trend, which needs to be interpreted cautiously. Additionally, as omega-3 was just one of a broader set of factors that were examined as potential moderators, there is a potential for Type 1 error given the multiple comparisons conducted. Replications in larger and more diverse samples to confirm and extend our preliminary findings are needed.

In conclusion, this study underscores the roles of environmental tobacco exposure and omega-3 consumption in shaping the persistence and change of childhood aggression. The significant interaction observed between environmental tobacco exposure and childhood aggression highlights tobacco exposure as a consistent risk factor across developmental stages. Conversely, the trend suggesting that frequent freshwater fish consumption may reduce aggressive behaviour highlight the dietary influences on behavioural outcomes. These findings suggest that interventions targeting parental smoking cessation and promoting healthy dietary habits, particularly omega-3 intake, could potentially mitigate the risk of persistent antisocial behaviours in children. Future studies are needed to further inform effective preventive strategies for promoting healthy behavioural development in children.

## Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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## Supporting Information

Additional supporting information can be found online in the Supporting Information section.