Author's response to reviews

Title: Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: a preliminary cross-sectional study

Authors:

Jennifer Downing (j.downing1@ljmu.ac.uk)
Mark A Bellis (m.a.bellis@ljmu.ac.uk)

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Author's response to reviews: see over
Response to Reviewers and Editorial Comments

Red = Changes to text
Italics = response to reviewers and editorial comments

Title: Early pubertal onset and its relationship with sexual risk taking, substance use and anti-social behaviour: a cross-sectional study

Reviewer 1: Steve Shoptaw

Reviewer's report
A1 A major weakness to the paper is that the inclusion of the Internet recruited sample introduces substantial biases by interfering with understanding who, exactly, the participants are. Just eyeballing Tables 1a and 1b, Female participants in the Internet dataset (compared to paper) were more likely to be older, have more childhood illnesses and be poorer growing up. Males in the Internet dataset (compared to paper) were more likely to be older, be gay/bisexual and have more childhood illnesses. These differences highlight a historical bias, which is recognized as an important factor in childhood obesity and early onset puberty.

We agree with this referee and editorial comments that sampling issues mean that this should not be seen as a paper identifying relationships that, as yet can be generalised. Instead we feel the study provides a novel exploration of adolescent behaviour with interesting results capable of stimulating and informing further studies. We have deliberately included two very different samples and believe one of the paper’s strengths is that many of the associations between peri-pubertal and adolescent variables and age of pubertal onset appear in both samples. We have edited the text in a number of places to make it clear that the samples are opportunistic, differ from each other and are not generalisable to wider populations. However, we have also stressed that they highlight the need to explore interesting (and we believe) important associations between rates of child development and adolescent behaviours.

A new Table 1 has been added to make comparisons clear between the two samples and we have identified in the text how differences between the two samples are handled. For both females and males paper and on line samples differed significantly by age, sexuality levels of childhood illness and socio-economic status (table 1). Consequently, in bivariate analyses on line and paper samples have been treated separately and in multivariate analyses collection method has been included as an independent variable. (p8)

We have included the word preliminary into the title to stress this is an initial examination of these relationships

We have edited the conclusions in the abstract to stress the hypothesis forming nature of the results. Results provide sufficient evidence for changes in age of pubertal onset to be further explored as a potential influence on trends in adolescent risk behaviours. (p2)

In the methods we have stressed that this is an initial examination of these relationships and that we were deliberately seeking different populations. As an initial exploration of relationships between early pubertal onset and adolescent risk behaviours, two different populations (one paper based and one online) were surveyed. (p7)

We have also identified some of the advantages of using two different samples in the discussion.
However, although our online and paper based samples differed significantly in age structure (table 1) both independently identified similar relationships between weight and early puberty in girls (table 2a) and early pubertal onset and alcohol and sex-related risk behaviours in both sexes (table 3a, b). (p11)

We have made it clear in the discussion that these results, while of public health interest, cannot be generalised to broader population effects.

These initial results are insufficient to establish population level effects but at least suggest that alcohol consumption, having been drunk, drug use and smoking under 14 years may be associated with earlier puberty in both sexes. (p11)

Further, we have already described that this study is limited to exploring associations and cannot measure effects sizes.

In light of the retrospective nature of this study, results here are insufficient to quantify any contribution that earlier puberty may be making to the relationship between these major public health issues and pubertal onset. However, our results suggest an urgent need to examine how diet and deprivation are affecting maturation and behavioural development in children and what measures can be put in place to reduce associated harms. (p11)

Finally, we have begun the conclusions with a clear statement regarding the limitations of this initial examination of these issues.

The opportunistic samples examined here were neither of sufficient size or demonstrably representative to extrapolate to broader populations. However, two different samples both provided some evidence of associations between early pubertal onset and major public health challenges facing a youth that physically develop at a substantially greater rate than 150 years ago [6]. (p14)

A2. The problem with these biases is magnified by repeated assertions that the two samples are considered as “independent” and as convenience samples, but the paper then makes sweeping generalizations/conclusions on health and social policy for maturing early adolescents. The biases inherent to these samples interfere with making any full-fledged generalizations or conclusions. The findings need to be constrained to guiding future confirmatory research.

As identified above we have now made it clear throughout the paper that these results are an initial examination and cannot be generalised. We have included statements expressly designed to clarify that we do not want readers to generalise from these results. However, they should simulate examination of other potential (here developmental) influences on adolescent behaviour and public health consequences of factors affecting age of pubertal onset.

These initial results are insufficient to establish population level effects but at least suggest that alcohol consumption, having been drunk, drug use and smoking under 14 years may be associated with earlier puberty in both sexes. (p11)

Such results at least suggest a hypothesis that earlier puberty is a contributor to, or a risk factor for, a greater propensity for violence. (p12)

Our results suggest that the occurrence (tables 2a, b) of early puberty should be explored as a potential factor influencing adolescent behaviour especially in deprived communities and as a possible contributor to health inequalities. (p13)
A3. It is disappointing that no hypotheses were proposed to guide the analyses, especially in light of the very thorough introduction section that set the stage for defining the variables measured. Providing some evidence showing expected (or by contrast, surprising) outcomes from the data analyses would help, especially since there are no family-wise alpha corrections made to reduce risks for Type I errors (which needs to be conducted in any revision of this report).

To clarify that we are working to a hypothesis that early pubertal onset is associated with greater adolescent risk behaviours we have changed the text in a number of places. We have also made it clear that our choice of variables against which we explore age of pubertal onset have been chosen through reference to existing literature.

In the background:
Consequently, here we retrospectively measure relationships between early onset puberty in UK males and females in order to examine whether earlier puberty is associated with greater participation in risk-taking behaviour during adolescence. (p5)

In the discussion:
However, we limited independent variables to key demographics and those factors previously identified as linked with age of puberty in other studies. (p11)

While we are aware of a long and on-going debate about family-wise alpha corrections we have now made it clear why we feel such corrections are not appropriate to this study. This paper is a novel exploration of associations between puberty and some key public health issues facing adolescents today. Our choice of variables was deliberate and directed by existing papers. Our choice of analyses appropriate given the associations we were exploring and the range and number of categories analysed relatively small. We feel that the risk of Type I errors is small and that the risk of Type II errors from applying corrections would be more damaging to our results helping steer future research. These arguments and discussions are well rehearsed in the literature and we have included appropriate references to allow readers to reach their own conclusions.

Finally, by exploring a range of factors potentially relating to early puberty and subsequent risk behaviours, there is a risk of Type I errors [40]. However, we limited independent variables to key demographics and those factors previously identified as linked with age of puberty in other studies. Importantly, with this study being an initial examination of potential public health consequences relating to earlier pubertal onset we opted not to include family-wise alpha corrections which would increase risks of type II errors [41,42]. (p11)

Minor Essential Revisions:
a. The number of male and female subjects for “age at survey” in Tables 1a and 1b do not equal the number of participants listed in the method section. As this would seem to be the most complete analysis, what happened to the missing participants? b. Along this line, please explain how missing data were managed.

We have clarified this in the text.
Sample sizes vary slightly between analyses where not all questions have been completed by all respondents. Sample sizes are included with each analysis (see tables 1, 2a, 2b and 3a, 3b) in order to clarify response rates and numbers being analysed. (p8)
c. It is not appropriate to label Table 1a as “Age of Menarche” and 1b as “Age of Puberty” given the variables in the column were the “independent (or predictor) variables.” The dependent or criterion variables were age of menarche and age of puberty (as noted in the table legends).

We have altered the tables in line with the referee’s request. (p26-27)

Reviewer: Muideen Bakare

Major Compulsory Revision

B1. One would notice that being overweight was identified to be predictive of early pubertal onset. I however, doubt the reliability of self assessment of weight status retrospectively as employed by this study. There are a number of confounding factors that may influence this, ranging from re-collection to self perception of what being overweight implied which may vary from one individual to another, especially in western culture that promote slimness as a measure of beauty. Therefore, apart from highlighting the limitations of retrospective design of the study, I would advise that the authors discuss separately the limitation of this retrospective self assessment of weight status as employed by this study.

We have now addressed issues of recall bias at a number of places in the text
Retrospective design also risked problems with passage of time confounding recall of other variables (e.g. self-assessed peri-pubertal weight, family structure, personal illness and substance use and sexual behaviours) and subsequently their relationships with pubertal onset. However, although our online and paper based samples differed significantly in age structure (table 1) both independently identified similar relationships between weight and early puberty in girls (table 2a) and early pubertal onset and alcohol and sex-related risk behaviours in both sexes (table 3a, b). (p11)

We have also highlighted the issues around recall and cultural issues affecting retrospective weight assessment and that despite these we have still seen similar patterns of association between weight and pubertal onset reported elsewhere. Despite retrospective reporting of weight problems being potentially prone to recall issues and variations in social norms, our results were largely consistent with studies examining pubertal onset and weight elsewhere [34]. (p13)

B2. The authors inferred that more childhood illnesses in females were also predictive of early pubertal onset in their samples, but they did not provide further information about what types of childhood illnesses they assessed. Was it just any childhood illness, whether chronic or acute illness? The nature of the illnesses assessed by the authors would offer further useful information.

We have clarified this in the text.
For the latter variable typical childhood illness were listed (i.e. measles, mumps, chicken pox, whooping cough, pneumonia) but individuals could also report any other illness they suffered during childhood and regarded as major (e.g. meningitis). (p5)

Minor Essential Revisions

a. Third sentence under “Results Section”:
Adding “-------------------------------younger age (at the time of survey)”, would make the statement to be clearer to the readers.

We have changed this as requested. (p2)
**Discretionary Revisions**

b. Reference numbers are better placed before the ‘period sign’ rather than after the ‘period sign’.

We have addressed this as requested.

**Comments from the Editor**

A major weakness to the paper is that the selection of the samples introduces substantial biases, in particular the inclusion of the Internet recruited as shown by the characteristics of this sample. In this respect, one of the referees introduces also the term of historical bias, which is appropriate. I have also concerns with the retrospective design, the low response rate, the definition of puberty you used and the confounders you adjusted on in the LR models.

In responses to comments to referee 1 (see A1) we have made substantial changes to the text to stress that this study is an initial investigation, that relationships are within individual from two populations providing results that should not be extrapolated to wider populations. We have however also highlighted how this is an important and relatively novel approach to understanding persistent public health issues relating to adolescent behaviour.

We have also addressed the difficulties in a retrospective design in number of places. Retrospective design also risked problems with passage of time confounding recall of other variables (e.g. self-assessed peri-pubertal weight, family structure, personal illness and substance use and sexual behaviours) and subsequently their relationships with pubertal onset. (p11)

We have also addressed these in relation to weight recall in particular (see response to referee’s comment B1).

Despite retrospective reporting of weight problems being potentially prone to recall issues and variations in social norms, our results were largely consistent with studies examining pubertal onset and weight elsewhere [8]. (p13)

We fully understand that a higher response rate would have reduced selection bias. However, we now have stressed that this initial study examines relationships within individual respondents and does not seek to extrapolate results to broader populations.

Both online and paper-based surveys were limited by opportunistic sampling and, combined with completion rates of around 60%, raise the probability of self-selection and sample bias. However, analyses only examined relationships between variables describing any individual and did not attempt to extrapolate to wider populations. (p10)

We feel that the paper has used well referenced and established mechanisms for establishing a proxy measures for pubertal onset in both girls and boys. While prospective studies may be better able to establish these, they would require long term follow up before puberty and through adolescence. We would be delighted to see such studies undertaken and one of the objectives of this initial work is to stimulate such work.

In discussion:

Although retrospective studies can be prone to errors in data recollection our median menarcheal age (13.0 years) is in line with other studies examining age at menarche in the UK (i.e. median age 12 years, 11 months) [4]. For males, physiological measures, such as spermate and facial hair growth, have been used as markers of puberty [38]. However, like other studies [15] we employed a range of behavioural markers as proxies for pubertal onset. Importantly, our proxy measures produced a median age of 11.0 years and while there are no comparable data from the UK this is consistent with studies elsewhere (USA; range: 10-12 years) [3]. (p11)
Finally, for the confounders in LR model we have deliberately included all demographic measures. We have now clarified this and the categorisation of these variables in the footnotes for both figures.

For all analyses, other than pubertal onset, independent variables included all key demographics measured: age at time of survey, socio-economic grouping, ethnicity, sexuality, and data collection method. The categories used for these variables are as described in table 1. (p29)