



# **Maternal Depression: Comments on Cummings & Kouros; Campbell; Goodman & Rouse; Toth & Peltz; and Cooper, Murray & Halligan<sup>1</sup>**

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## **Topic**

*Maternal depression*

## **Introduction**

Target reviews in this section, written by leading investigators, offer brief, authoritative accounts of the key research findings in this area and how they might help to influence policy and practice. They do this by articulating a range of clinical research questions, and findings, with sizable implications for the affected individual and family and for society as a whole. There is substantial agreement across reviews concerning what is known and what is not yet clear. Moreover, there is agreement on how these authors conceptualize the task of understanding the impact of maternal depression with how investigators in other areas of behavioural science struggle to understand the impact of risk exposures. Accordingly, if we can manage to resolve some of the practical and strategic issues raised in these reviews – pinpointing the source of individual differences in risk exposure, integrating biological with psychological models and measures, translating scientific understanding to improve detection and treatment – then we will have made an advance that will generalize to other multi-determined clinical problems.

## **Research and Conclusions**

These reports say little that is disagreeable; more positively, they highlight important lessons for this particular area and for developmental psychological research more broadly. Toth & Peltz's<sup>1</sup> emphasis on the need to account for the multiple biological factors is characteristic of contemporary research protocols; Goodman & Rouse's<sup>2</sup> attention to mechanisms and questions of timing of risk exposure typifies leading research programs on a wide range of topics; Campbell's<sup>3</sup> focus on parenting (shared by other authors) highlights the practical need for theory and clinical intervention to target specific mediators of risk exposure; Cummings & Kouros<sup>4</sup> document the tendency of research studies to over-focus on a particular risk for child outcomes without considering

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<sup>1</sup> Comments on original papers by Cummings & Kouros and Toth & Peltz published in 2009, and by Campbell, Goodman & Rouse and Cooper, Murray & Halligan published in 2010. If these versions are no longer available online and you wish to have access to them, contact us at [cedje-ceecd@umontreal.ca](mailto:cedje-ceecd@umontreal.ca).

the broader family context; Cooper and colleagues<sup>5</sup> highlight the advantages that treatment studies provide over observational research designs, and how findings from treatment studies pose further challenges for theory and methods.

The reviews identify findings worth reiterating and expanding, namely, a) the concept of developmental timing, b) challenges in inferring causal connections, c) the need to incorporate biological models.

*a) Developmental timing.* One finding requiring further consideration is that the effect (or presumed effect) of maternal depression on the child is evident even in infancy. That may be a consequence of early appearing genetically-mediated effects (see below). Whether or not genetic factors are at play, however, this observation implies that comprehensive research studies need to begin early in development. Studies starting early in the child's development would, for example, make it possible to track the unfolding developmental dialogue between child and parent that each author discussed or alluded to. In addition, studies including the period of early infancy and childhood would permit a test of whether or not infants are especially sensitive to the effects of disrupted early care, such as often accompanies maternal depression. Experimental studies on animals<sup>6</sup> demonstrate that this is so, and there is a clear need to examine if that extends to humans. Importantly, the suggestion that studies commence in early infancy follows not from the evidence that there are particular effects of maternal depression in infancy, but rather from the need to test that possibility. For example, in the case of maternal postpartum depression, the case for early exposure as having particular impact is far from certain.<sup>7,8</sup>

There are, in fact, many problems that derive from not knowing enough about the role of developmental timing – the hypothesis that there are certain periods in the child's development when she/he is particularly vulnerable to the impact of maternal depression. Many studies contribute provocative findings but are unable to resolve this debate. For example, if the effects of maternal depression are observable in the first months of age, then one might wonder if parental caregiving quality were a likely causal factor (i.e., is it likely that the impact of parental care would have taken hold on the child's development by that point? We don't yet know.). A practical expression of this scientific question is whether or not there are optimal periods to intervene. For example, interventions for depressed mothers with young children have been undertaken, and may result in improvements in child-parent attachment.<sup>9</sup> So far as we can tell, there does not yet seem to be a point (e.g., child age) after which interventions for parent-child relationships or child outcomes are demonstrably ineffective.

*b) Causal inference.* Exposure to maternal depression will almost certainly mean exposure to a host of other factors that will also compromise child development (even if they were not combined with maternal depression); many of these are noted in the target articles. Maternal depression may therefore be conceptualized as a proxy variable, as with poverty, parental divorce/remarriage, and many other risks that dominate research on child development and psychopathology. Indeed, given that, by its nature, maternal depression increases the risk for other adversities (and results from other adversities), it is reductionist to account for a specific effect of maternal depression. That does not mean that maternal depression is not a worthy target for research or treatment, but rather that

observational studies simply do not have adequate leverage for disentangling inherently tangled effects.

One example of this is paternal depression. There is some suggestion in the reviews that paternal depression has been ignored; the effect of that is potentially severe. Assortative mating for psychiatric disorder – the tendency for women with psychological disorder to pair with men with psychological disorder and vice versa – is well-known and confounds research and clinical efforts focusing on only one or the other partner. Moreover, a recent study showed that even in the postpartum period, a period of risk conventionally ascribed only to women, the risk for depression is elevated in men.<sup>10</sup> A separate study demonstrated that paternal postpartum depression predicted child adjustment problems independently of maternal postpartum depression.<sup>11</sup> Nevertheless, most studies of maternal postpartum depression make no mention of paternal postpartum depression, or consider the broader family setting.

The intervention study design should be seen as especially valuable because it offers far greater leverage for testing causal hypotheses. For example, several studies<sup>12,13</sup> suggest that changes in maternal depression (induced through intervention) had positive “down stream” effects on child outcomes; that implies a far firmer link than associations obtained from observational studies.

*c) Biological models.* Many contributors noted that an exclusive focus on behavioural outcomes in the child is limiting. If there are behavioural effects, then there must also be biological effects. There is at present no compelling organizational model that would point to one or other focus for biologically-focused research on the impact of maternal depression, but many candidates exist, as noted. A recent longitudinal study is notable because it expanded research on maternal symptoms well beyond behavioural outcomes and their underlying causes. That study linked maternal symptoms with illness and specific markers of immune functioning in the child.<sup>14</sup> If parental depression could be linked in a causal way to child’s immunological or cardiovascular health (and we are not there yet), then the public health impact of parental depression would be substantial, and addressing parental depression would then be seen as a focus for all health care providers rather than just those in mental health.

Given the brevity of the reports, it would be inevitable that some important features would be left out or go undeveloped. The most obvious of these is genetics. Many contributors mention genetic factors, but the practical application of this is not particularly straight-forward. Twin and adoption studies provide some additional leverage for detecting genotype-environment interplay. Molecular genetic studies are arguably easier and cheaper to perform, although the challenge here is in knowing what the specific risk *allele* is doing, even if something is detected. And, there is not yet robust evidence that genetic factors predict treatment response. These are important caveats for future research. Nonetheless, it is instructive that genetic effects are reliably included in contemporary studies. Relevant studies show that genetic factors may be associated with parenting behaviours.<sup>15</sup> and that parenting effects on the child may depend on the child’s genetic make-up.<sup>16</sup> That complements the extensive evidence that maternal depression is under some degree of genetic control. It would be unusual if the pervasive (although

perhaps not extensive) role of genetics as described above were not evident in the causal chain linking maternal depression to child outcomes.

### **Implications for Development and Policy**

One of the more important but most complex implications for policy concerns the extent of individual differences in human development. Simply put, there is wide variation in children's responses to stresses, even where there is a focus on a specific exposure, such as maternal depression, and even when that exposure is extreme, as in the case of institutional rearing.<sup>17</sup> That can be seen in the current collection of papers in the authors' efforts to qualify most findings that were reported, and allude to such factors as children's temperament, cognitive sophistication or any of a number of other factors that might influence why some children may respond worse than others. Even if maternal depression were a monolithic exposure – and surely it is not – the effects on children would be diverse because of the varying cognitive, genetic, and other sources of vulnerabilities in the children.

Wide variation in outcomes in children exposed to a particular risk implies that there will be wide variation in response to any particular evidence-based intervention – whether it is family therapy or home visiting. That is why there is so much focus in intervention studies on the question, “what works for whom?”, or what is referred to in the research literature as “moderators” of treatment outcome. The explicit message is that no particular intervention will work for everyone; not all participants will respond clinically to evidence-based interventions. That presents a complex and perhaps unwelcome story that is difficult to convey swiftly to a lay audience, and it is, of course, awkward for moving forward on policy. We need to be prepared for the inevitable finding that an intervention (clinical or policy) is certain to have varying effects – it may be brilliant for some, irrelevant for others, and possibly counter-productive in still others. It may be that the movement toward individualized medicine, which follows naturally and logically from genetic research, will provide a metric for targeting and tailoring interventions. But, we are not there yet. Flexibility in treatment and in policy, for example, with regard to a range of possible interventions, is a key take-home message.

A second implication is the need to focus on population-level estimates of risk and adjustment, a topic not much covered in these reviews. Unfortunately, research shows how difficult population-level change is to achieve, and the number of studies that qualify as informative in this regard is small.<sup>18</sup> Furthermore, despite a concerted research efforts for many years, there has not been a documented drop in maternal depression or child adjustment problems; in contrast, claims have been made about an epidemic of childhood depression, although that is not based on sound evidence.<sup>19</sup> A goal of policy may therefore be to commission efforts to understand how successes in individual treatment studies and advances in research do not make healthier populations.

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