The other day I saw a new mom with a 5-week-old baby who spent a good part of his day crying. The mom had done the things moms with babies who cry a lot often do: she had blamed something in her breast milk and stopped breastfeeding, then she had started working her way through the various infant formulas in the grocery store; still her baby would not stop crying. “He looks like he’s in pain. He scrunches up his little fists.”

Findings of the baby’s examination were reassuring, and he was gaining weight appropriately. I told her everything pointed toward colic as the diagnosis. I explained that, with the notable exception of shaken baby syndrome, colic usually has a good outcome for babies, moms, and the baby-mom dyad. A family member had moved in for a time to help with the baby, and the mom was happy she had someone to whom she could hand him off when she felt herself becoming helpless or angry. She asked if she should continue with the probiotic drops she had been giving the baby; I said there was not much evidence it would help with her son’s crying but that there was likely no harm in trying.

Usually my colic visits end around there, with reassurance and arrangements for follow-up. It can be a dissatisfying exchange for both patient and physician: simple reassurance that “nothing is wrong” can be mistaken for minimizing, and it also does not necessarily help a patient with her very real distress. On bad days, it feels intellectually fraudulent: if nothing is wrong, then what evidence do I have that it is “right”? The causes of things

As a mother of 3 kids younger than 7, I would be the last with the probiotic drops she had been giving the baby; I what evidence do I have that it is “right”? But lately I have been wondering what might happen if medicine were to focus less on the familiar right-wrong and good-bad dichotomies, and invest more in wonderings about the causes of things. Not just proximate questions such as “What might a parent have done to make her baby cry right now?” but the chewier, ultimate question: “Why should a baby cry?”

This is the question that intrigues Dr Ronald Barr, a pediatrician at the University of British Columbia in Vancouver and author of the website The Period of PURPLE Crying. At a recent workshop I attended at Simon Fraser University in Burnaby, BC, on the evolutionary aspects of child development and health, Barr explained how he had used the methods of anthropology and the theoretical framework of evolution to generate 2 hypotheses. His first was that colic—excessive infant crying that peaks around 6 weeks of age and resolves by 3 months of age—is costly but is not explained by pathology and it should be reconceptualized as normal. Barr compared infant crying and soothing behaviour within Western populations, and also between Western populations and the !Kung San people of Botswana, who follow a seminomadic subsistence lifestyle. In spite of very different circumstances, he found that babies in all cultures had similar frequencies of crying, and also that other early infant behaviour—namely, turning toward a rattle, caloric intake, and habituation to visual stimuli—had similar n-shaped curves reflective of increased responsiveness during the first 3 months of life. Put another way, excessive infant crying might not be “wrong” or “bad” or even indicative of a problem; rather, it could just represent a normal variant of infant development.

Barr’s second hypothesis was that colic might in fact be adaptive and consistent with an honest signal mechanism. Strictly speaking, a trait is considered “adaptive” in evolutionary terms if it improves an individual’s chance of running the natural selection gauntlet and passing on her genes to the next generation. This definition is quite narrow in the context of contemporary humans, though, who interfere with natural selection in any number of ways, such as practising contraception, performing cardiac surgery, and paving over the habitat of large predators.

Measuring adaptation in modern humans relies on subtler measures of resilience, such as allostatic load (the toll stress takes on the body) and chronic low-grade inflammation. To understand honest signaling, think of a peacock or a stag: the most robust males make the brightest plumage and the largest antlers. Although one might imagine that infant crying is a nonadaptive trait because it increases the allostatic load of the mother and erodes her ability to provide for her child, it is also possible that, according to honest signaling, it is the healthiest babies that can afford the cost of crying.

Barr’s approach to a clinical question really fired me up as someone who had studied evolutionary biology before medical school. It begged so many questions: Is colic neutral in terms of adaptive advantage, therefore...
not subjected to selective pressures and thus persisting because it has no effect on survivorship? Not likely, because crying is costly in terms of energy demand on the baby and also the devastating outcomes of shaken baby syndrome. Is genetic control of colic linked to some adaptive trait, so that colic has simply hitched a ride? Is colic the result of a mismatch between past and present environments, much like the way we think hypertension might be a consequence of our ancestors having lived in the context of salt-poor diets? Or might colic in fact be adaptive? Is a crying baby picked up more and subsequently fed more or better protected from sabre-toothed tigers?

These questions are interesting in their own right. But what was more powerful to me as a primary care provider was how Barr used his data not to ask how we might change infant behaviour, but to question our assumptions about—and our interventions in—infant behaviour. After discussing all of this with my patient, a wave of relief passed over her face. “So it might not be my fault?” Her baby’s crying had been distressing to her, but what weighed more heavily on her than the actual screaming was the “common knowledge” that babies cry only from distress and she must be doing something wrong as a mother.

**Informing clinical care**

This reformulation of colic is just one example of how the insights of evolutionary biology applied to medicine—*evolutionary medicine* for short—stand to transform both our clinical care as family physicians and medical research in general. Take, for example, the work of Dr Thomas McDade, an anthropologist at Northwestern University in Evanston, Ill. Drawing on the Cebu Longitudinal Health and Nutritional Survey, which recruited 3000 pregnant women from the second-largest urban area of the Philippines in the 1980s, McDade and colleagues have been able to determine that exposure to microbes during infancy (eg, increased diarrheal morbidity and exposure to animal feces around the home) correlates with lower levels of C-reactive protein (CRP) in adulthood, and that women with lower levels of CRP during pregnancy— even before they are pregnant—have higher-birth-weight babies. This is important because the Barker hypothesis tells us that people with low birth weight are at greater risk of chronic diseases such as cardiovascular disease and diabetes. Also, parental absence before the age of 11, as when a mother leaves to work abroad, was a significant predictor (P < .05) of high CRP in adulthood (and therefore lower-birth-weight babies at higher risk of chronic disease in the next generation), but only for kids who were raised in microbe-deficient environments. The mechanism McDade proposes to explain this is the following: in a high microbial environment, a child has multiple opportunities to turn inflammation on and off; when that child grows to adulthood and is exposed to a pro-inflammatory trigger—anything from an infection to psychosocial stress—he or she can more efficiently turn inflammation off once it is no longer needed.

In addition to begging many more research questions—Do kids born on farms in Canada have less cardiovascular disease? Does separation from parents contribute to cardiovascular disease burden in refugee populations or kids in government care? Should we expect impaired glucose metabolism to be a sequela of the new nature deficit disorder?—McDade’s findings also raise potential challenges to current clinical guidelines. Should I be measuring CRP in all of my prenatal patients, and should an intervention be designed for those patients with high values? Should we tell new parents to throw out their hand sanitizer?

I am not ready to do either (just yet), but these questions relate to my original example of how evolutionary thinking can inform clinical care. At the end of the first day of the workshop, a provocative discussion facilitator asked everyone in the room to think about the purpose of evolutionary medicine. It seemed to be a no-brainer for most participants, by far most of whom were basic scientists rather than physicians. I also thought it was a no-brainer for me: these lines of research will lead to new and better treatments and reduce human suffering, of course! But when I really thought about it, the answers I came up with were more complex.

An evolutionary perspective could reframe illness experiences for individuals and help improve their self-efficacy. New moms’ breast milk has been taking a couple of days to “come in” for a really long time now, and yet breastfed babies usually thrive. An evolutionary timeline can go a long way toward normalizing certain behaviour. In a similar vein, we doctors are fond of “educating the patient” as a means of managing the so-called diseases of lifestyle. Some of my most clean-living patients are the first people in their families to have high cholesterol. An evolutionary perspective might help shift secondary prevention away from name-and-blame to a consideration of developmental aspects of disease and the interaction between genes and environment.

An evolutionary perspective might also help temper clinical hubris by adding a long look toward future generations. Everything is a trade-off, and we do not always fully understand the long-term implications of our interventions. Does treating a woman’s iron deficiency anemia in pregnancy predispose her to bacterial infections, resulting in lower-birth-weight babies with higher cardiovascular disease risk? Does intrapartum prophylactic treatment of group B streptococcal colonization predispose affected infants to asthma or obesity?
Finally, an evolutionary perspective might provide additional justification for population health measures. By protecting the health of moms and young women today, we might be reducing the disease burden of at least the next generation as well. That is powerful medicine.

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Competing interests
None declared

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