

One possible explanation is that G-actin binding to MAL in the nucleus does not permit assembly of an effective transcription complex. Serum response factor lies at the nexus of two major signaling pathways that control the expression of different genes. One pathway involves the signaling enzyme MAP kinase and the activation of transcription coactivators of the ternary complex factor family (3). The other pathway relies on the myocardin family of coactivators (to which MAL belongs) (4). Because both families of coactivators bind to the same region of serum response factor, the G-actin–MAL complex may have reduced affinity for serum response factor. Another possible explanation relates to chromatin regulation by G-actin. In yeast and mammalian cells, G-actin is present in a number of protein complexes that remodel chromatin (5), enhancing adenosine triphosphatase activity of these complexes (6). It has been estimated that 10% of total nuclear G-actin is associated with SWI/SNF-like BAF chromatin-remodeling complexes. G-actin in these complexes may interact with MAL, physically linking MAL function to that of the remodeling complexes (inhibiting transcription), presumably by forming chromatin structures that repress gene expression. However, MAL has not been found associ-

ated with chromatin-remodeling complexes, making this explanation less attractive. A more likely explanation of how transcription by serum response factor is blocked by the MAL–G-actin complex is that G-actin simply interferes with MAL association with components of the general transcription apparatus, thus preventing serum response factor from activating transcription.

Skeptics might argue that many mechanisms elaborated in cultured cells may only be true of the cell line, with its specific chromosomal breakages, DNA methylation patterns, and thereby altered genetic circuits. This is almost certainly not the case with the work by Vartiainen *et al.* Although the authors used fibroblast cell lines, aspects of their mechanism are supported by rigorous genetic studies in mice. For example, deletion of the *serum response factor* gene in mice leads to death of embryos at gastrulation (7), when both transcription and actin-induced cell movement are essential. Conditional deletion of *serum response factor* in the murine nervous system produces specific defects in neurite outgrowth and neuron migration that are linked to reduced expression of actin and its regulators (8, 9). Finally, mice genetically engineered to lack *MAL* have defects in myoepithelial cell differentiation (10).

Cell biologists have long thought of actin regulation in the context of controlling cell morphology and movement. However, if confirmed by additional genetic studies (for example, analysis of mice with mutations in *MAL* that block its interaction with actin), the work by Vartiainen *et al.* elucidates how actin choreographs the regulation of morphology and transcription. Such a coordinated genetic circuitry must underlie such diverse events as early embryonic development, neuron migration, blood vessel formation, and lymphocyte signaling.

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10.1126/science.1145014

PSYCHOLOGY

Birth Order and Intelligence

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Research on birth order and intellectual performance is replete with contradictory findings and long-standing conceptual disagreements. In the wake of these ongoing controversies, a new study that has profited from past debates is especially welcome. In an elegantly designed analysis of 241,310 Norwegian 18- and 19-year-olds that appears on page 1717 of this issue, Kristensen and Bjerkedal show that older siblings have higher intelligence test scores than younger siblings (1). In addition, these two researchers demonstrate that how study participants were raised, not how they were born, is what actually influences their IQs.

In a companion study, Bjerkedal *et al.* (2) show that birth-order differences in their Norwegian sample are nearly identical for a

subset of adjacent siblings who were raised together (127,902 individuals) and for a between-family sample (112,799 individuals). Critics have long argued that such birth-order effects, which typically emerge in between-family studies, are spurious—phantom artifacts of uncontrolled differences in family size, socioeconomic status, parental IQ, and other background factors (3–5). At least in the domain of intellectual ability, the new Norwegian findings rule out this alternative explanation.

Critics might still argue that the mean IQ difference documented between a Norwegian firstborn and a secondborn is only 2.3 points. Such a modest difference, however, can have far greater consequences than most people realize. For example, if Norway's educational system had only two colleges—a more prestigious institution for students with IQs above the mean, and a less desirable institution for all other students—an eldest child would be

After nearly a century of debate, a large study shows that birth order influences intelligence, but the reasons remain to be resolved.

about 13% more likely than a secondborn to be admitted to the better institution (the relative risk ratio), and the odds of a firstborn being admitted would be 1.3 times as great. In medicine, new therapeutic benefits of this magnitude often make front-page headlines. In addition, such differences in opportunities gained or lost inevitably accumulate over one's lifetime.

One puzzle highlighted by these latest findings is why certain other within-family studies have failed to show equally consistent results. Some of these previous null findings, which have all been obtained in much smaller samples, may be explained by inadequate statistical power, as Bjerkedal *et al.* themselves suggest. But most previous researchers have overlooked another intriguing reason for such inconsistent outcomes, which are generally found in studies of children rather than adults. As has been noted by Zajonc and colleagues, younger siblings tend to score higher than

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IQ-related effects	Confluence theory	Resource dilution theories	Prenatal theories	Spurious association theories
1. Birth-order differences in within-family data	✓	✓	✓	✗*
2. Functional versus biological birth-order differences**	✓	✓	✗	✗
3. Family-size effects	✓	✓	✓	✓
4. Only-child deficit	✓	✓	✗	✓
5. Twin deficit	✓	✓	✓	✗
6. Birth-order differences dissipate with large age spacings	✓	✓	✗	✗
7. Reversal of birth-order differences by age	✓	✗	✗	✗

*Such explanations predict limited (but spurious) birth-order differences in between-family data only.
 **As demonstrated by Kristensen and Bjerkedal in this week's issue.

Sibling differences. The efficacy of models predicting differences in intellectual performance. Classifications are based on whether each theory offers a possible explanation for reported IQ differences, although not necessarily a correct explanation.

older siblings when tests of intellectual ability are conducted under the age of about 12 (6, 7). In more than 50 previous samples, there is a significant tendency for IQ disparities by birth order to reverse direction as children get older.

Zajonc's own confluence model of intellectual ability provides a possible explanation for this curious age-related reversal in birth-order effects. According to this model, the family's overall intellectual environment embodies a dynamic aspect that includes all of its members' relative contributions. For example, the intellectual environment of a firstborn at, say, age 7 is actually less favorable than the environment of a 2-year-younger sibling at the same age. This is because the younger sibling, being linguistically and cognitively less mature, degrades the firstborn's intellectual environment, whereas the older sibling enriches the secondborn's environment. To explain why older siblings eventually tend to overtake their younger siblings in intellectual performance, Zajonc's model posits a tutoring effect, which kicks in as older siblings begin to teach what they know to their younger brothers and sisters. Through the organization and expression of thoughts, teaching younger siblings is posited to benefit the tutor more than the learner, especially since lastborns have no one to tutor.

Given the latest findings from Norway, it is useful to compare the features of various competing theories about birth order and intelligence and to assess how they now stack up. These alternative explanations include family resource dilution models (of which the confluence model is a sophisticated variant), theories about prenatal influences, and "admixture" theories asserting that birth-order effects are spurious products of uncontrolled confounding influences. As shown in the table,

resource dilution models and the confluence model both do well in providing possible explanations for birth-order differences, as well as for other family-related effects in intelligence (8). For example, both models are consistent with the fact that children without siblings, who are more likely than other children to grow up in single-parent homes and who also lack a sibling to tutor, generally exhibit lower test scores than firstborns having a younger sibling. Similarly, twins are expected to score lower than singletons, either because of gestational factors (twins compete for resources inside the womb) or because they dilute the family's intellectual environment more than do singletons. Without going into further detail about the relative merits of the various models outlined in the table, it is nevertheless noteworthy that only the confluence model addresses the apparent reversal in intellectual performance by birth order as children are growing up.

The confluence model has been criticized repeatedly over the past three decades (4, 5). Although this embattled model has survived these critiques, it is not without unresolved problems. One difficulty is the absence of any direct evidence showing that tutoring by older siblings actually raises their IQs, although indirect evidence is suggestive (9). A plausible alternative to the supposed effects of tutoring involves competitive niche partitioning within the family. Well-designed within-family studies have consistently shown that firstborns are rated by themselves, their parents, and their siblings as being more self-disciplined, hard-working, and intelligent than their younger siblings, and also as being "the achievers" of the family (10–12). Although such perceived sibling differences might well reflect differences in family roles, or even sib-

ling stereotypes, rather than real or permanent differences in personality or ability, such competitive role differentiations and shared beliefs may also help to explain why elder siblings, by early adulthood, have higher IQs than their younger siblings.

Thanks to the new results, we no longer need to wait for truly persuasive data to justify those theories that consider birth-order differences in intellectual performance to be a within-family phenomenon. It seems likely, however, that portions of past theories—formerly uncompromising rivals—may be required with-

in any theory that is adequate to the task of explaining findings from large national samples. For example, parents who tend to have small families may, on average, have higher IQs than do other parents, contributing to family-size effects in both between- and within-family data (5). Similarly, gestational factors may no longer provide a plausible explanation for birth-order effects relating to intelligence, but they are still relevant to understanding why twins have lower IQs than singletons. The greatest challenge that now confronts birth-order researchers is to find, and to creatively mine, other large data sets like that available in Norway, so that alternative explanations can be tested against one another, allowing some of these adversarial rivals a continuing, if more restricted, role in a multifaceted explanation.

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