

Dear Dr. Rubenstein:

This is a reply to your letter of March 17, 2002. The letter you sent was a reply to Dr. Galves' letter of February 16, 2002 in which he expressed concern about the following three statements that are included in the brochures on ADHD that were published by Division 29 (Psychotherapy) in conjunction with Celltech Pharmaceuticals Inc:

1. ***“ADD/ADHD is generally considered a neuro-chemical disorder.”***
2. ***“Most people with ADD/ADHD are born with the disorder, though it may not be recognized until adulthood.”***
3. ***“ADHD is not caused by poor parenting, a difficult family environment, poor teaching or inadequate nutrition.”***

Dr. Galves' basic objection to these statements was that there is no scientific evidence to support them.

In your letter to Dr. Galves, you included information and references that were provided to you by Dr. Robert J. Resnick and Dr. Kalman Heller. We have reviewed the information and references you sent and continue to find that they do not contain scientific evidence for the statements in question. We submit to you the following analysis of the information and references you provided as well as additional information and references.

“ADD/ADHD is generally considered a neuro-chemical disorder”

Although ADD/ADHD may be generally considered by popular opinion to be a 'neuro-chemical disorder,' there is no scientific evidence to back this claim. The

scientific evidence to which you refer contains only equivocal and inconsistent evidence that the brain physiology of individuals diagnosed with ADD/ADHD is different from that of individuals not diagnosed with the disorder (Goldstein and Goldstein, 1998; Barkley, 1990; Ross and Ross, 1982). Even if there were more solid and conclusive evidence it would not support your implication that ADD/ADHD is **caused** by these biological dynamics.

All that we can derive from our careful review of the literature you cite is that there is evidence of a *correlation* between the biological dynamics and the ADHD category. Because this evidence is entirely correlational and the brain is a living, functioning organ constantly responding to its environment with complex neurochemical and other neurofunctional changes, it is just as likely (and perhaps more likely) that the biological dynamics are a **result** of an interplay of emotions, thoughts, intentions and behavior experienced by the diagnosed individuals. Please consider the following research findings in relation to this perspective:

- Jeffrey Schwartz et.al of UCLA (1996) found that a group of people suffering from obsessive-compulsive disorder had “abnormalities” in their brains. Half of the group received drug therapy; the other half received cognitive behavioral “talk therapy.” All of the patients improved and, when Schwartz checked their brains, he found that their brains had changed in the same ways. Presumably, the cognitive-behavioral therapy had the same impact on the physiology of the brain as did the biological therapy.

- Mark Rozensweig et.al. (1972) found that the brains of monkeys raised in rich environments had a greater number of neurons and more complex interneuronal connections than the brains of monkeys raised in more impoverished environments.
- Franz Alexander (1984) found the people who had been deprived of support, affirmation and ample time while growing up were much more likely to suffer from overactive thyroids than people who were brought up in more nourishing environments.
- James Pennebaker (2000) found that students who were assigned the task of writing about traumas they had suffered and about their fears, relationships and desires had stronger immune systems and were healthier than students who were assigned to write about less emotionally charged topics.
- Studies have demonstrated a relationship between vulnerability to depression and the following psychological variables:
 - Suffered trauma at an early age (Kramer, 1993);
 - Have a high need for and/or lost an important relationship (Johnson and Roberts, 1995);
 - Use a ruminating style of thinking (Lehmicke & Hicks, 1995);
 - Score low on self-esteem and high on stress (Kreger, 1995);

- Have lost control over important variables in their life (Jensen, Cardello & Baun, 1996);
 - Hold a stable rather than flexible attributional style (Seligman, 1975).
 - Score high on a scale of self-defeating personality (McCutcheon, 1995).
- A recent study by Cornell researchers found that a two-week course in remedial reading significantly changed the brain physiology of dyslexic students (Rappaport, 2003).
- A recent study by Seattle psychiatrist Arif Khan (Khan et al., 2002) indicated a large overlap in effect between placebo and antidepressants in the original FDA trials of these drugs. Leuchter and fellow UCLA researchers (Leuchter et al., 2002) found that these placebo effects result in detectable changes in brain function. Similar studies have *not* been undertaken with ADD/ADHD subjects *nor are individuals who may have already experienced temporary or permanent brain changes as a result of stimulant treatment typically excluded or controlled for in ADD/ADHD research* (Leo & Cohen, 2002).
- Baumeister and Hawkins (2001) undertook an exhaustive review of efforts to substantiate a neuroanatomical site or sites related with ADD/ADHD through structural and functional neuroimaging techniques such as PET, single positron scanning, MRI, and

electrophysiological measurement. These researchers stated that while “[t]here seems to be a consensus among experts today that ADHD is associated with structural and/or functional abnormalities in the brain”, they could only conclude that “the present review indicates that the neuroimaging literature provides no convincing evidence for the existence of abnormality in the brains of persons with ADHD” (p. 7-8).

The above evidence contradicts your premises. The scientific principle of parsimony compels us to arrive at a completely different set of conclusions than you have—in which the biological dynamics you cite as correlating with ADD/ADHD at the brain level **can be more accurately depicted as a result of psychological and environmental variables** than a neurodevelopmentally damaged, diseased, or dysfunctional brain. The mind-body dynamic that has been most thoroughly researched in this regard is the human stress response. The human stress response is a profound, complex biochemical and physiological dynamic that is preceded by a perception of threat and a cognition that the threat is real and needs to be dealt with. The psychological variables of the human stress response precede and likely cause the physiological variables, rather than the converse (Everly, 1989; Selye, 1974).

Calling ADHD a “neurochemical disorder” with a “biological cause” implies that it has nothing to do with how a child thinks, feels, reacts, intends, perceives, adjusts and responds. It implies that the behaviors are not under the control of the child or those within the child’s world and have nothing to do with how the child finds and makes

meaning in that world. That is a fundamental error contradicted by those of us who, like you, also work very closely with children and families everyday.

“Most people with ADD/ADHD are born with the disorder, though it may not be recognized until adulthood.”

The implication here is that ADD/ADHD is a genetic disorder. There is a body of research that purports to demonstrate that this disorder is essentially a result of genetic factors. Most of that research has used studies that compare interclass correlations between the rates of the disorder in monozygotic twins and dizygotic twins. Virtually all of this research has found significantly higher correlations between monozygotic twins than between dizygotic twins (Goodman & Stevenson, 1989; Pauls, 1991; Biederman et al., 1992; Gillis et al., 1992; Edelbrock et al., 1995; Sherman et al., 1997). However, this kind of research suffers from the following serious deficiencies:

- All of this research is based on the assumption that monozygotic twins and dizygotic twins are raised in equivalent environments.

That assumption is erroneous. As Jay Joseph (2003) has explained:

Identical twins spend more time together than fraternal twins, and more often dress alike, study together, have the same close friends and attend social events together. James Shields, in his 1954 study of normal twin school-children found that 47% of the identical twins had a ‘very close attachment’ which was true for only 15% of fraternal twins . . . According to Kringlen’s (1967) survey, 91% of identical twins experienced ‘identity confusion in childhood’ which was true for only 10% of fraternal twins. Kringlen also found that identical twins were more likely to have been considered as alike as two drops of water (76% vs. 0%), ‘brought up as a unit’ (72% vs. 19% and ‘inseparable as children’ (73% vs. 19%). Sixty-five percent of identical twins were found to have an

‘extremely strong’ level of closeness which was true for only 19% of the fraternal pairs (p.).

Since the equal environment assumption is not valid, the correlations between monozygotic twins are just as likely a result of environmental factors as of genetic factors.

- Findings of genetic influence over behavior are confounded by the fact that genes direct the synthesis of protein and protein synthesis can be affected by environmental factors such as stress, trauma and lack of parental responsiveness (Hubbard & Wald, 1993). The process of gene expression is much more complex than is suggested by stories in the popular press (Commoner, 2002). Thus, the process through which genes influence the behavioral characteristics of a person is itself greatly influenced by environmental factors.
- In order to scientifically demonstrate genetic etiology for any trait, the precise genetic mechanism involved must be identified. As Ross and Ross (1982) point out:

The only procedures that can precisely define a genetic mechanism are segregation studies which could only be done with humans under very unusual circumstances and linkage studies which would require the identification of the genetic marker associated with hyperactivity....and these are possibilities for which there is as yet no evidence.” (p. 73, 74)

These flaws cast doubt on the validity of the research that purports to show a genetic etiology for ADD/ADHD. Even without considering these powerful contaminating factors and obstacles, the research on genetic factors in ADHD accounts for no more than

50 % of the variance. This is hardly a reasonable basis for your declaration that ADD/ADHD is present at birth.

A second approach to demonstrating genetic etiology is by using research on the correlation between infant temperament (Thomas and Chess, 1977) and later diagnosis of ADD/ADHD. Some theorists have suggested that such temperament factors as activity level, threshold of responsiveness, intensity of reaction, distractibility and attention span and persistence of these elements might be associated with characteristics of behavior disorders such as ADD/ADHD later on. Thomas and Chess (1977) indicated, for example, that “features of temperament played *significant roles* [emphasis ours] in development of childhood behavior disorders.” However, those same researchers concluded that, “in no case did a given pattern of temperament, as such, *result in* [emphasis ours] behavioral disturbance. Deviant development was always the result of the *interaction* between a child’s individual makeup and significant features of the environment.” (p. 40). Indeed, the most carefully administered study of this factor found that “the contributions of family characteristics and pre-natal/perinatal characteristics are outweighed by the contribution of constitutional factors (hyperactivity in the family, chronic illness as a child and temperament characteristics) *and by the home environment domain (measures of achievement press, provision of early learning activities and parent-child interactions)* [emphasis ours]” (Lambert & Harsough, 1984)

A third approach to inferring genetic etiology of ADHD is research that compares the incidence of ADHD and other psychiatric disorders in the relatives of children diagnosed with ADHD with the incidence of such disorders in relatives of children not diagnosed with ADHD (Safer, 1973; Biederman et al., 1986; Pauls, 1991). This research

is confounded by the failure to control for the many environmental factors that could also explain the intergenerational transmission of mental disorders in families. Research on attachment dynamics and trauma demonstrate the profound influence that parent-child relationships in the first months of life have on the mental health of individuals. (Holmes, 1995; Bretherton, 1995; Crittenden, 1995; Lewis, Amini & Lannon, 2000; Herman, 2000; van der Kolk, McFarlane & Weisath, 1996). None of the research on the incidence of ADHD in families controls for these crucial factors.

Research and common sense confirm that genetic inheritance must have some influence over temperament and, therefore, over the behaviors that characterize ADHD. However, research also demonstrates that genetic influence is not a major factor. As three psychiatrists Lewis, Amini and Lannon (2000) put it:

Genetic information lays down the brain's basic macro-and microanatomy; experience then narrows still-expansive possibilities into an outcome. Out of many, several; out of several, one.....While genes are pivotal in establishing some aspects of emotionality, experience plays a central role in turning genes on and off. DNA is not the heart's destiny; the genetic lottery may determine the cards in your deck, but experience deals the hand you can play.....Like most of their toys, children arrive with considerable assembly required...A child's brain cannot develop normally without the coordinating influence that limbic communication furnishes. The coos and burbles that infants and parents exchange, the cuddling, rocking, and joyous peering into each other's faces look innocuous if not inane; one would not suspect a life-shaping process in the offing. But from their first encounter, parents guide the neurodevelopment of the baby they engage with. In his primal years, they mold a child's inherited emotional brain into the neural core of the self." (, pp. 149-153)

A balanced review of this research indicates that there is no scientific evidence that ADD/ADHD is present at birth as you have claimed and that genetic factors are, at best, a minor influence over the behaviors that characterize ADD/ADHD.

“ADHD is not caused by poor parenting, a difficult family environment, poor teaching or inadequate nutrition.”

In fact, a preponderance of the scientific evidence demonstrates that ADHD is *significantly associated* with unmet needs for nurturance in childhood, difficult family environments and inhumane and oppressive school and community environments. Researchers have found an association between the behavioral characteristics of ADHD and the following characteristics of parenting and family environments:

- Family instability, differences in press for achievement in the family, provision for early learning, disciplinary practices, interest in the child’s schooling, negative and pessimistic perception by parents of the child’s academic and intellectual competencies accompanied by decreased expectation levels and decreased desire to participate with the child in learning activities. (Lambert and Harsough, 1984)
- Parents feeling threatened and inadequate; parents unconsciously rejecting the child and parents blaming children for the extra problems they present. (Lambert, 1982)
- Mothers’ use of criticism and general malaise in parenting. (Goodman and Stevenson, 1989).
- Father’s hypercritical and destructive attitude, inconsistent, impatient and pressuring parenting approach and mothers who are judged to be emotionally disturbed (Thomas and Chess, 1977).
- Maternal anxiety and attitude toward pregnancy (Sameroff & Chandler, 1975).

- Mothers who are more directive commanding and negative; parents with depression, alcoholism, conduct disorder, anti-social behavior and learning disabilities; mothers who are less responsive to positive or neutral communications of their children (Barkley, 1990)
- A negative, critical and commanding style of child management (Campbell, 1990)
- Parental distress, hostility and marital discord (Cameron, 1977)
- Greater familial anger during conflicts, more disengagement from each other and repeated disputes over school issues and issues pertaining to siblings; parents who adhered to rigid beliefs about their teens' bids for autonomy and who attributed misbehavior to malicious intentions (Robin, Kraus, Koepke and Robin, 1987)
- Parents who use aggressive behavior, indiscriminate aversiveness and submissiveness or acquiescence toward their children during management encounters (Patterson, 1982).
- Disharmony in early mother-child relationships (Battle and Lacy, 1972).
- Experiences of high level of stress in parenting and feelings of lower self esteem (Goldstein and Goldstein, 1990)
- Mothers who were critical of their difficult babies during infancy and showed lack of affection for them continued to be disapproving and tended to use severe penalties for disobedience during the primary school years and assessed their children's intelligence as low (Ross and Ross, 1982).

The authors and the research you cite fail to account for two rich areas of research that have clearly demonstrated the impact of early familial experience on the behaviors characteristic of ADHD: *attachment and trauma*.

Attachment researchers have found significant relationships between the quality of mother (and father) – child relationships in the first months of life, the quality of attachment (secure, disorganized or avoidant) at one year of age and the school performance, sociability, levels of anxiety and general health of children in primary and secondary school (Goldberg, Muir and Kerr, 1995). As J. Holmes (1995) puts it, “Attachment research has shown that a school-age child’s sense of security is greatly influenced by the consistency, responsiveness and attunement he or she experienced with his or her parents in infancy.” Certainly, the behavior that is used to diagnose ADHD can be seen as the normal and understandable reaction of an insecure child to a stressful situation.

Researchers who have studied trauma have found that traumatic experiences early in life have a great impact on the ability of victims to modulate their emotions and to react effectively and appropriately to stressful and frustrating experiences (van der Kolk, McFarlane & Weisaeth, 1996; Herman, 2000). Trauma victims tend to become easily activated by threat and adversity, to react impulsively; or they protect themselves by shutting down and retreating into themselves. Both of these are behaviors that are used to diagnose ADHD. Traumatic experiences do not have to be life-threatening to have such an impact. They can consist of deficits in love, support, nourishment, affirmation that are experienced as being life threatening.

Deutsch et al. (1982) found that adopted children are much more likely to be diagnosed with ADHD than non-adopted children. This is understandable in view of the fact that all adopted children have suffered the trauma of being ripped away from their birth mothers.

Your inattention to the two rich lodes of research regarding attachment and trauma in relation to early experience and the kinds of behavior used to diagnose ADHD are major deficiencies in the research that you cite.

The brochure also denies the impact of “poor teaching” on ADHD. While ‘poor teaching’ may, indeed, not be ‘to blame’ for the rise of ADD/ADHD, the inhumane, oppressive, absolutely stultifying environment of the typical public school as a primary factor is undeniable. Current educational curriculums appear designed to be stuffed down the passively-receptive throats of students through repetitive, boring worksheets, one-size-fits-all, standardized methodologies, and minimal or no opportunity for active learning. Seldom is a child asked what he or she wants to learn or how she or he wants to learn it. Children are subjected to a horribly skewed value system in which primary emphasis is placed on linguistic and mathematical intelligence at the expense of other intelligences that are just as important: musical, spatial, mechanical, kinesthetic, interpersonal and intrapersonal. If children become bored, frustrated, and complain about it, they are told to be quiet or go to the principal’s office. Worse than this, these children may be shuffled into the special education diagnostic category of ADD/ADHD and placed in ‘less over-stimulating’ classrooms. In such circumstances, it is the children who are now pathologized as the ‘problem’ and ‘abnormality’ rather than a major societal system that fails to serve them.

Many scholars have testified to the ways in which the typical school hurts children by failing to encourage them to develop into the unique, separate, creative beings they crave to be (Leonard, 1968; Holt, 2000; Gatto, 2001). Others have noted that ADHD is diagnosed by watching the behavior of children in a typical classroom and that, if you put those same children in a less oppressive environment, they don't engage in such behaviors. So Alfie Kohn (2000) wonders if we are diagnosing the child or the learning environment. And Willerman (1973) asks, "Should we classify a high level of activity and a low tolerance for being forced to pay attention to something one doesn't want to pay attention to as a disorder?"

Even the ADHD researchers you have cited have found evidence of the school environment's impact on diagnosis of ADHD:

- Inattention is most dramatically seen in situations requiring the child to sustain attention on dull, boring, repetitive tasks in which there are minimal immediate consequences of completion (Barkley, 1990)
- Task failure or a sudden reduction in anticipated reward or reinforcing feedback may severely disrupt behavior (Barkley, 1990);
- Pre-school hyperactive children were notably more restless, difficult and off-task than their nonhyperactive peers when required to engage in academic-type pursuits such as sitting at a table and listening but were indistinguishable from their peers in free play (Ross & Ross, 1982);
- Onset of hyperactivity often coincides with the point of school entry (Ross and Ross, 1982);

- Hyperactive children perform best on self-paced tasks and their behavior often deteriorates on ‘other-paced’ tasks (Ross & Ross, 1982);
- Hyperactive children have a difficult time in school, particularly in adolescence, when school work becomes more demanding and achievement becomes an important goal--this situation improves in adulthood when they can select for themselves a job in which they can succeed. (Ross and Ross, 1982).

Are we diagnosing a child or are we diagnosing a learning environment that is intolerable and damaging to a particular cohort of children with certain characteristics who are then called ‘mentally ill’ (ADD/ADHD) only because some of us choose to call them that?

We can think of many reasons why a child would resist being forced to pay attention to something that doesn’t meet his or her need or that diverts him or her from something that is considered more important at that moment:

- She may have some deep concerns that are so troubling that she doesn’t have space for anything else:

Will I ever have any friends that I can really depend on and feel safe with?

Is there something I can do to help my parents be happier so they can do a better job of nurturing me?

Why is it that I have so much trouble doing this work and the other kids seem to be able to do it with ease?

- He may have a burning desire to express a talent or drive that is not being honored. When he was ten years old, Picasso’s teachers were concerned because all he wanted to do was paint.

For practitioners of professional psychology to treat such concerns as a ‘mental illness’ and respond with a ‘prescribing predisposition’ is a disservice to a child whose individual crisis needs to be understood and used as an opportunity for learning—not how to read, write and do math but how to manage his emotions, thoughts and intentions and how to get along with other children without losing himself.

That ADD/ADHD is generally considered to be a neuro-chemical, genetic disorder with little relationship to parenting and environment is a case of popular opinion being at odds with scientific evidence.

What are the implications of ‘buying into’ such popular opinion?

This is, unfortunately, not a new dilemma for our discipline. As thousands of ADD/ADHD evaluations continue to be undertaken by psychologists nationally, we would do well to recollect the early days of applied clinical psychology when culturally biased IQ testing of immigrants, African Americans and Native Americans was used to bolster conclusions regarding the genetic inheritance of “feeble-mindedness” on behalf of the American eugenics social movement. At that time, many psychologists were just as convinced of their methods and theories as many continue to be about ADHD currently. In fact, no less than six presidents of the American Psychological Association signed up in some advisory capacity with eugenics organizations and initiatives over a twenty-year period. That is about the same time period with which we have witnessed our field involved with ADHD.

It was only tolerance for a diversity of views and a critical minority of applied psychologists of that time that helped to gradually extricate our field from a morass of significant racist biases. There are recognized scholars inside and outside APA who would submit that the current ADHD diagnostic descriptor, as well as many others contained within the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), was composed in a controversial manner (Caplan,1996) and that ADHD itself has been substantiated only through poorly conceived and implemented research procedures using instruments of questionable reliability and validity (Carey, 1998; Armstrong, 1997).

The rise in popularity of the ADHD diagnostic category appears to be occurring on behalf of an American cultural movement relevant to the rearing of children that arises from changing values, mores, and demands in work and family life, educational curriculum, media exposure, and perception of time and time management. We should be vigilant of the multitude of voices and perspectives relevant to these changes, rather than lending our credibility to a single perspective that disregards or minimizes such factors in favor of a suspect reductionist, biological explanation.

Let our letter sound a warning to you that much is at stake here. As the ADHD category has already begun to be exported from its current white, middle-class, male youth focus to children of color, who continue to grow up under conditions of abject poverty and oppression, the die is being cast for psychology's complicity in fostering a new, modern class of eugenics survivors – the ADHD child of color, shuffled to special education rooms as an individual “behavior issue”. Thus, we rationalize away our failure to accurately identify and effectively address his or her problem as having a primary

origin in inequity, injustice, poor parenting and the failures of American public education.

The matter we are discussing here is of the utmost importance to psychology and to the people who are treated by psychologists. If we see the hyperactivity, impulsivity and “disinhibition” that characterize ADHD as driven by genetics and random biological dynamics, we call it a disorder and treat it with drugs and techniques of operant conditioning. If we see that same behavior as a functional response of the child to a situation that is difficult, off-putting, oppressive, abusive, irrelevant, discounting, disaffirming, and/or inhumane, we can call it a normal and understandable reaction and treat it by helping the child, family, and caretakers to fashion a better, more adaptive and life-enhancing response.

What does it imply if psychologists such as ourselves do not agree as to the existence of biological or neurological causes of ADD/ADHD? What does it imply if they have witnessed misuse and abuse of the category of ADHD with defiant, traumatized, or disruptive children of color? What does it imply if psychologists question the reliability and validity of their own diagnostic procedures with respect to ADHD?

It does not mean that children are not having trouble sitting still or paying attention to teachers or caretakers.

All we wish to demonstrate is that there is no “true ADHD” but only debatable ADHD. And we wish to emphasize that you are creating an American Psychological Association brochure and not a brochure for the American Psychiatric Association. There are many clinicians who do not subscribe to the reductionist, medical model implied in your efforts to legitimize ADHD as a so-called “neuro-chemical disorder.” They have

weighed the same evidence you have and have come to contrary conclusions. We submit that the American Psychological Association has an obligation to present a more balanced account of the professional views of its constituents. That includes yours—and ours.

Many of the statements you make in your letter are far from conclusive to the APA practitioners you are attempting to represent in the brochure program. How can we hand our clients any brochure that seems to favor the zealotry of biological psychiatric views about ADHD as somehow unquestionably true when this is far from so, while minimizing and even invalidating the value of psychosocial explanations we might offer? This is disenfranchisement of our particular perspective on ADHD from our own professional organization.

We ask that Division 29 immediately cease the distribution of these brochures and that other brochures be produced that reflect a more balanced account of the available scientific evidence and the wide diversity of views of practicing psychologists regarding ADHD. As the most visible national public body of psychologists, we strongly recommend that:

- o We indicate in these brochures that neurobiological explanations for ADHD are based on limited and controversial research findings
- o Professional psychologists hold a variety of perspectives and opinions about the diagnostic category of ADHD and its etiology
- o ‘Best practices’ of professional psychologists serving children, youth, adults, and families in relation to the descriptor of ADHD will vary in their approach based on what makes sense to each of us as professionals,

what we know about human beings, and what appears to be in the best interests of all our clients

- o As a body of practicing psychologists, we acknowledge before the public and one another that what we believe about ADHD is based on neither adequate nor established scientific fact but is instead a reflection of cultural and societal forces that have influenced our theoretical, research, professional, and practicing agendas
- o We should publicly urge all psychologists to keep an open mind as we continue to work on the controversies we have raised surrounding the ADHD issue together.

We submit this letter to you in the spirit of collaboration and consultation. Although some of our differences with your perspective are great, your views have challenged us and inspired deeper thought as we clarify our own for you here. For that, we are very grateful to you.

Sincerely,

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David Cohen, Ph.D.	Barry Duncan, Ph.D.
Michaele Dunlap, Psy.D.	Harris Friedman, Ph.D.
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Bertram P. Karon, Ph.D.	Kirk J. Schneider, Ph.D.
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Have you guys found any solution? For me changing editor to MonoDevelop and back to Visual Studio is not working. Click to expand Old thread but it doesn't seem to have the answer I needed and it seems to happen on every new script I add with VS open at the time. What I did that has worked so far was to close and reopen VS, this gets my new script in the Solution Explorer. Then I click the Refresh button at the top of the solutions explorer in VS to correct the problem with monobehaviour not being inherited and made my intellisense work again. bglassmaker_unity, Oct 21, 2017. #10. recognize the characters. maybe they are in the book, but MAYBE they are in the page source. Right click and select "View Page Source":