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ESSAY

Seeking Immortality? Challenging the Drug-Based Medical Paradigm

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Introduction to Henry Bauer—I am very honored this morning to introduce the recipient of the 2012 Tim Dinsdale Award, Professor Henry H. Bauer. I trust that everyone in this room is aware of the enormous contributions that Henry has made to Anomalistics and to science in general. He was not only one of the Founders of the Society for Scientific Exploration, but also one of its greatest contributors. We owe him very much.

Let me say a few words about his background. Henry was born in Vienna in 1931, but went through school and university in Sydney, Australia. After a Bachelor of Science (given with Honors) he proceeded to receive a Master of Science and Doctorate from the University of Sydney, which then kept him on for seven years to teach in the Department of Agricultural Chemistry. After that he taught at the University of Kentucky for another twelve years, and then went to Virginia Tech, where he spent 21 years before retirement in 1999, and where he is still Professor Emeritus. He was Dean of Arts and Sciences at Virginia Tech for eight years, an experience that led him to write (under a pseudonym) a book called *To Rise Above Principle*. In addition to his regular appointments, he had visiting appointments at the University of Michigan, the University of Southampton, and Rikagaku Kenkyusho in Tokyo.

I will not dwell on his many contributions to electrochemistry, but will simply note that he was author or co-author of three books and many articles and presentations on that subject. Already by the 1970s, however, Henry had begun to turn his attention to science studies, and in particular to the role that anomalies and unconventional science played in scientific thought. This led to research and eventually to books on Immanuel Velikovsky and the Loch Ness "Monsters" and most recently to a strongly dissenting book on the relationship of HIV and AIDS. In addition he wrote several books on the social relations of science, including one with the intriguing title *Scientific Literacy and the Myth of the Scientific Method*. Although a complete gentleman in all his activities, Henry never shrank from confrontation with and opposition to what he considered bad science. I think the phrase "uncompromising integrity" is appropriate here.

> I need to note that Henry has written yet another book, which will be available shortly: *Dogmatism in Science and Medicine: How Dominant Theories Monopolize Research and Stifle the Search for Truth*.
>
> And finally, in awarding Henry this Dinsdale Prize, it is impossible not to underline the singular appropriateness of this prize, since he is not only, like Tim Dinsdale, the author of a book on Loch Ness, but in 1978 was made an Honored Companion of the Loch Ness Explorers. And finally I might add that it gives me special pleasure to give this award to a very good friend.
>
> I give you Henry Bauer. Professor Bauer, would you stand and receive the citation for this award? —Ron Westrum

I appreciate and value this honor more than I can find words to say. This Society has been my intellectual home, and to be recognized in this way by this group is meaningful beyond words.

By serendipity or synchronicity, what I've been looking into recently happens to fit perfectly into the theme of this year's conference, challenging mainstream paradigms. My challenge is to the routine treatment of chronic ailments with drugs, which focuses on symptoms rather than causes and reflects an implicit, unwitting presumption that lifespan can be extended indefinitely.

Some of the following assertions may seem excessively iconoclastic (I'll be rather disappointed if they don't), but everything is based solidly on mainstream sources that have not been seriously questioned. I will give here just a few citations on critical points, but all the factual claims can be verified in a rather lengthy bibliography available at my website (Bauer 2012).

When we don't feel well, it's broadly speaking for one of two quite distinct reasons: Either we have contracted an infection, in other words we've been attacked from outside by a bacterium or a virus or a parasite; or we are experiencing a non-infectious, internally generated problem. Some of those are temporary, and my concern here is with those that are chronic rather than episodic—cardiovascular disease, for example. A chronic ailment bespeaks some disturbance of the intricately interlocking, system-regulating processes of signals, reactions, and feedbacks that normally keep us functioning properly—the phenomenon of homeostasis, physiology keeping itself stable.

Since illnesses arise in these two quite different ways, it seems on first principles that they need to be addressed in quite different ways. Instead, present-day practices apply the same approach in both cases, namely, treatment with drugs.

That's a plausible response to attacks from outside by infectious diseases, and indeed those have been largely overcome by antibiotics,

substances able to kill intruders without causing intolerable collateral damage to ourselves. The success of drug treatment of infections is owing largely to the facts that intruders are a specific identifiable cause of illness; the physiologies of bacteria and of parasites are sufficiently different from ours that chemicals can be found that kill the intruders with considerable selectivity and minimum damage to ourselves; treatment can be of quite short duration, days or a few weeks, so that collateral damage done by the drugs is not so serious that it cannot be repaired. Such repair is often needed, for instance because antibiotics also kill the beneficial bacteria in our digestive tracts which are the immune system's first line of defense.

By contrast, treating non-infectious disease, chronic disease, with drugs means lifelong consumption of medication and the accumulation of damage from "side" effects. Yet this is the standard approach nowadays to conditions described as chronic diseases.

With infectious diseases effectively under control, medicine became increasingly preoccupied with the main non-infectious causes of death—the failure of organs and rogue mutation of cells to generate cancers. These are malfunctions for which no single specific cause has been found. Indeed, as a matter of empirical fact, the unsuccessful decades-long search for causes suggests that there is no single one. Cancers are presently thought to be caused by a series of necessary but individually insufficient steps; even when one of them appears to be particularly effective, it is still not in itself decisive: For example, no one disputes that smoking plays a major role in the incidence of lung cancer—speaking statistically, that is; but smoking is neither a necessary nor a sufficient cause of lung cancer, for some people who have never inhaled tobacco smoke contract lung cancer while some heavy smokers live to the ripest old age. When it comes to ailments of heart and arteries, some 243 "risk factors" have been reported (Institute of Medicine 2010).

It would seem clearly irrational to imagine that conditions with such a range of partial apparent causes could be rectified by administering a few drugs, yet that is the current practice: Cardiovascular disease is treated with a variety of cholesterol-lowering drugs, blood-pressure–lowering drugs, blood thinners, beta-blockers, and more.

Now I was actually wrong to say that these non-infectious conditions, chronic and not episodic conditions, have no single specific cause. They do have a single cause. They are brought on by getting older. When someone dies from heart disease or cancer, it's regarded quite properly as death from natural causes. So present-day medical practice prescribes drugs as though that could stave off or even reverse what is a normal progression. The implicit but unacknowledged ambition is to prevent aging.

This ambition—one might call it hubris or chutzpah—is facilitated and masked by semantics: Conditions of aging are talked of as though they were illnesses; normal changes with age are labeled **ab**normal. The ubiquitous term *cardiovascular disease* implies that this is similar to other diseases including infectious diseases. "Hypertension," "too high," obviously describes an abnormal state. "High cholesterol" again implies something abnormal, unnatural, undesirable, *sick*—and "bad cholesterol" sounds even worse and more unnatural. "Dysfunction," as in erectile, means not working properly. "Pre-diabetes" suggests that you will later become ill; yet the actual fact is just that the level of blood sugar under some circumstances happens to be a bit higher than is average for some population of young, healthy individuals.

So chronic diseases are at least partly created or invented by definition. Further, because there is no single identifiable cause and since the symptoms are those of natural aging, taking drugs to treat or prevent chronic ailments—organ failures, cancers—seems very unlikely to work. But that is just arguing from first principles. Since drugs have actually been used to treat chronic diseases for several decades, there ought to be empirical evidence with which to check this a priori reasoning.

There is not.

Has it been the case that statins or blood thinners or anti-hypertensive drugs have prolonged life, with acceptable quality of that prolonged life?

There are no conclusive data-based answers.

The problem is that the proper question—the effect of treatment on all-cause mortality and quality of life—could only be answered by literally impossible studies. For each condition and each treatment of it, one would need to follow for decades a large number of individuals in two groups, a treated group and an untreated control group, the two groups being matched individual by individual for every variable that might affect the condition and its outcome—age, sex, genes, environment, lifestyle including diet.

Such trials being impossible, evidence has been sought indirectly and circumstantially via *biomarkers* or *surrogate markers*: Measurable characteristics whose magnitudes are taken to reflect the status of the condition being treated. For instance, as measures of cardiovascular disease (or risk of it), commonly used biomarkers include blood pressure, various cholesterol-related numbers, proteins associated with inflammation, and blood-clotting time.

But how do we know that any given surrogate marker actually reflects the state of the so-called disease?

We don't.

To know that a surrogate marker is inextricably linked with whatever

actually *causes* a particular ailment would require one of those literally impossible clinical trials. Surrogate markers are chosen because they've been found to be associated in some manner with a particular ailment. Surrogate markers are correlated with the condition they purportedly measure; but correlation never proves causation.

The incidence of organ failures and of cancers increases with age. Mortality increases with age. Blood pressure increases with age. The incidence of strokes and of heart attacks increases with age. As we get older, hearing deteriorates. The incidence of dementia increases with age. The incidence of type-2 diabetes increases with age. Arthritis becomes more common as we age.

Everything that increases with age correlates inevitably with everything else that increases with age. Such correlations do not demonstrate that one factor causes the other. It is worth always bearing in mind the example given by Huff (1954) in *How to Lie with Statistics*: Over many years, there was a perfect correlation between the salaries of Protestant ministers in New England and the price of Cuban rum. Did this mean that ministers were receiving salary raises so that they could afford to consume Cuban rum? Or was the ability of the ministers to pay more leading to greater demand for Cuban rum and therefore higher prices?

Of course not. Everything that has a dollar value attached to it experiences increases over time because of inflation, and so—*mutatis mutandis*—the dollar values of many things correlate with one another. But those correlations do not prove that one thing is the cause of the other.

Blood pressure correlates with organ failure, stroke, heart attack, cancer, arthritis, erectile dysfunction, hearing loss, dementia—among other things. That does not demonstrate that high blood pressure is a perfect marker for any of those things, still less that it actually causes any of those things. Correlation never proves causation. Yet present-day medical practice is to administer pressure-lowering medication when blood pressure exceeds a level that has been designated as "hypertension"—designated arbitrarily, based on opinion that mistakes correlation for causation.

Some months ago an entrepreneurial research group reported a correlation between loss of hearing and dementia. Shortly after that my local paper had a full-page advertisement in which a group of audiologists urged everyone to have their hearing tested and if necessary to get hearing aids in order to stave off Alzheimer's (Beltone 2012):

> If hearing loss goes untreated, a condition called "auditory deprivation" occurs, this has been confirmed by scientific studies. Seniors with hearing loss are significantly more likely to develop dementia over time than those

> who retain their hearing, a study by Johns Hopkins and the National Institute on Aging researchers suggests. The findings, researchers say, could lead to new ways to combat dementia, a condition that affects millions of people worldwide.
>
> Although the reason for the link between the two conditions is unknown, the investigators suggest that a common pathology may underlie both or that the strain of decoding sounds over the years may overwhelm the brains of people with hearing loss, leaving them more vulnerable to dementia. They also speculate that hearing loss could lead to dementia by making individuals more socially isolated, a known risk factor for dementia and other cognitive disorders.
>
> But there's good news! Whatever the cause, the scientists report, their finding may offer a starting point for interventions—even as simple as hearing aids—that could delay or prevent dementia by improving patients' hearing. (www.hopkinsmedicine.org)

That may strike you as absurd. It should. Yet the evidence that high blood pressure causes cardiovascular disease is of exactly the same ilk as the evidence that hearing loss causes dementia—or for that matter, that dementia causes hearing loss. It is also worth noting that these absurd speculations came from researchers at Johns Hopkins University and the National Institute on Aging. Incompetence in statistics is pervasive, including in medical matters. Note too the suggestion that "a common pathology" may underlie both dementia and hearing loss: Yes, a common factor does underlie them, but it's not a pathology, it's just the process of getting older.

Since surrogate markers are correlations and not proven causes, the question becomes how valid a measure they might be of the chronic diseases with which they are associated. An Institute of Medicine (2010) report has the explicit aim of defining conditions for use of surrogate markers in order to limit the potential damage from using markers that are not strictly valid.

The report finds that even the most widely accepted surrogates are far from universally valid. Thus tumor size is *not* a valid measure of cancer progression or guide to prognosis—even as it is commonly used as such a measure. Blood pressure and cholesterol, commonly used to indicate risk or progression or prognosis of heart disease are not valid measures of heart disease. A recent article in the *British Medical Journal* says all this quite plainly: "There are no valid data on the effectiveness" of "statins, antihypertensives, and bisphosphanates" (the last are prescribed against osteoporosis) (Järvinen et al. 2011).

So the contemporary paradigm of drug-based treatment of chronic ailments is flawed in a variety of interlocking ways. The theoretical basis is misguided:

Aging is treated as an illness.
Illness is measured by surrogate markers that are not valid.

In application of that paradigm there are a number of technical flaws as well:

Drugs are approved on the basis of surrogate markers that are not valid; and there are a number of other flaws in the approval process.
It's wrongly presumed that drugs can be specific.
There are many deficiencies in clinical trials.
Statistical analyses are often made incorrectly.

The approval of drugs by the Food and Drug Administration (FDA) relies on data from surrogate markers; and the FDA requires only two statistically significant trials of at least 6 months duration as supporting evidence. This procedure was originally introduced as "accelerated approval" during the panicked early days of the AIDS era, when activists were clamoring for treatment. An integral requirement of the new procedure: There was to be monitoring of the actual effect of treatment once a drug came into general use. However, that requirement has not been fulfilled, for about three-quarters of all drugs. That is owing in some part to the fact that the costs of the approval process are borne by the company making the request for approval, and the law specifying such payments precludes the FDA from using those funds for post-approval monitoring.

Altogether, there is no system in place to ensure that adverse "side" effects of drugs are actually reported, let alone promptly; and there exist no specific guidelines about when a drug should be withdrawn from the market. Physicians and hospitals are requested to alert the manufacturers or the FDA of adverse events, but it is not a formal requirement and there are no guidelines or specified criteria to help doctors determine what should be reported. In any given case of a patient's condition worsening, the physician may be uncertain whether this can be clearly ascribed to the medication or should be interpreted as a failure of the medication to defeat the illness, which would seem the natural immediate presumption. Even when side effects are reported to manufacturers, there is no clearly specified obligation on the manufacturer to alert the FDA. The Adverse Events Reporting System of the FDA is estimated to pick up only about 10 percent of actually occurring negative side effects, in other words perhaps 90 percent of all adverse drug reactions go unreported (Bowser 2011).

In addition to the reliance on invalid surrogate markers, there are quite a number of reasons why clinical trials have not delivered reliable knowledge about the efficacy and safety of drugs used in the manner in which they are actually prescribed:

Conflicts of interest may bias the initial trials. Clinical trials are increasingly being done by Contract Research Organizations (CROs) (Mirowski 2011). Their business is more likely to thrive if their clients get the results they want. Predisposition on the part of drug companies and of CROs to expect that a new drug will be effective can influence how trials are carried out and interpreted.

Positive results of the initial clinical trials—on which approval is based—are often not reproducible. Of the 35 most highly cited studies based on biomarkers, only 15 of the tested drugs later achieved even nominal statistical significance in actual use, and only 7 of them had certifiably significant effectiveness (Ioannidis & Panagiotou 2011). That may be owing in part to biased clinical trials, but, further, drug companies are not required to submit the data from *all* trials, only from 2 successful ones; so there may well have been some that failed to deliver significantly positive results. At any rate: Experience shows that *80% of drugs (28 of 35) were approved on the basis of bad data that did not later hold up*.

Trials are not representative of who will be treated in practice.

1. The obvious way to test a drug is with the most clearly ill people. But after a drug has been approved, it will be used with people who are less ill. Drugs to lower blood pressure might be tested with people at Stage 2 or Stage 3 hypertension—above 160 or 180 systolic—and yet after approval those drugs will be prescribed for people whose pressures may be as low as 140 or 130 or even lower.

An actual example: Bisphosphanates achieved a 32% reduction in hip fractures among women aged 65–80 with actual osteoporosis or previous fractures. But if these drugs were administered to all older people at risk of osteoporosis, which corresponds to present-day practices, fewer than 5% of hip fractures would be prevented (Järvinen et al. 2011). Whereas a 32% reduction might make for a good benefit/risk ratio, a 5% reduction would be unlikely to do so.

2. "Off-label" uses of drugs are common. Doctors are permitted to prescribe a drug for any condition at all, not just those for which the FDA approved that drug. About 20% of all prescriptions in the USA were for off-label purposes—about 30% with psychotropic drugs (estimated cost, $26 billion), for instance the antipsychotic Risperdal was used off-label twice as often as on-label. Nearly half of the prescriptions for asthma medicines were off-label. Coumadin (warfarin) is commonly prescribed for hypertension and coronary heart disease even though never approved for such treatment. A review (Radley et al. 2006) found that about three-quarters of off-label applications had little or no scientific support.

Drug companies are forbidden from advertising off-label uses, but

they find ways of doing so nevertheless. GlaxoSmithKline agreed to pay $3 billion for illegally marketing Advair, whose annual sales approach $8 billion (Whalen 2012). For illegally promoting off-label uses, Pfizer had been fined $2.3 billion, Eli Lilly $1.4 billion, AstraZeneca $520 million, Bristol-Myers Squibb $515 million, and Novartis $420 million (Pharmalot 2010).

The record suggests that all the leading pharmaceutical companies find it profitable to break the law and pay the fines as just another cost of doing business. Not just by promoting off-label uses: Johnson & Johnson were fined $1.1 billion for hiding risks associated with Risperdal (Health Notebook 2012).

But what has been the human cost of innumerable individuals imbibing inappropriate medications? How much does this illegal quackery contribute to the overall costs of "health care"?

A host of deficiencies have to do with statistics:

Misguided or misleading interpretation: Correlations are persistently taken as demonstrating causes. Risk factors are confused with actual risks. A review in 2011 reported that half of all articles in top-ranked journals—*Science*, *Nature*, *Nature Neuroscience*, *Neuron*, and *Journal of Neuroscience*—used incorrect statistical procedures (Nieuwenhuis et al. 2011). Douglas Altman (1994, 2002) at the Center for Statistics in Medicine in Britain has published about this for more than two decades.

Data mining: Correlations are looked for in existing sets of data—recall the triumphantly published finding of the link between dementia and hearing loss, a perfectly predictable and uninteresting result since both increase with age. If one tries enough combinations, some purely chance associations will appear to be "statistically significant"—under the usual criterion of $p \leq 0.05$, on average at least 1 in every 20 apparent correlations will be spurious (and the other 19 should not be interpreted as reflecting a cause-and-effect relation).

Relative risks are reported instead of absolute risks. If A is a cause of death at a rate of 4 per 100, and if a treatment reduces the rate to 2 per 100, drug companies and the media will typically report a 50% reduction in risk, which seems eminently worthwhile. They might even put it that without the drug, the risk of death is 100% higher. But actually the risk of death is lowered by only 2 per 100, 2%, and that may *not* represent a good bargain with the side effects of any possible treatment.

Even beyond all that, I. J. Good or R. A. J. Matthews (1998, 1999) would point out that the standard frequentist method used in medical statistics (and in social-science research), taking $p \leq 0.05$ as a criterion, can deliver quite

misleading results, overestimating the actual significance of any results.

A consequence of accelerated approval and associated flaws is that an increasing number of approved drugs have had to be withdrawn again after less and less time on the market, because of dangerous side effects that had not been noted in the short clinical trials. For example, the anti-diabetic Rezulin, approved in 1997, was withdrawn after only 3 years; the antibiotic Raxar, approved in 1997, withdrawn after only 2 years; the appetite suppressor Redux, approved in 1996, withdrawn after 21 months; the calcium-channel blocker Posicor, approved in 1997 and prescribed against hypertension and angina, was withdrawn after 12 months; the analgesic Duract, approved in 1997, was withdrawn after 11 months; Lotronex, for gastrointestinal disorders, was approved in 2000 but lasted only 10 months before being withdrawn. The Wikipedia list of withdrawn drugs has 2 from the 1950s–1960s, 2 from the 1970s, and 5 from the 1980s. Following the introduction of accelerated approval, 15 had to be withdrawn in the 1990s and 26 in the 2000s.

Current practice is far too cavalier about so-called "side" effects. There is copious evidence that drugs approved for one purpose—reducing blood pressure, say—are likely to have undesired other effects. One illustration of the ubiquity of side effects is the drug industry's continuous efforts to "reposition" drugs, to have them approved for a different purpose than the original one. Purely commercial reasons underlie this practice. Once a drug has been approved, its safety has been established to the satisfaction of the FDA (which, as just pointed out, does not mean the drug is actually safe, however). That hurdle does not have to be overcome again when approval is later sought for some other use of the drug (Ashburn & Thor 2004). Special conferences (Drug Repositioning 2012) reflect the presently high interest in repositioning or "repurposing" drugs.

One example of multiple functions is Propagest, which used to be prescribed for nasal congestion, control of urinary incontinence, priapism, and obesity (Allergy Clinic no date). Since 2005, the Food and Drug Administration has been urging that the drug be withdrawn because of apparent association with increased risk of hemorrhagic stroke, showing the drug's effect on yet another aspect of physiology.

A second example of multiple functions, and still being prescribed, is Cymbalta, originally approved to treat anxiety, depression, diabetic peripheral neuropathy, and fibromyalgia (remarkably enough, all at the same dosage), clear enough evidence in itself that the substance affects a whole range of bodily functions. The drug was later repurposed successfully—under a new name, Duloxetine SUI—as treatment for stress urinary incontinence.

Even more worth pondering than multiple applications is that a given drug may exert opposite side effects in different people at different times: Side effects noted with Cymbalta/Duloxetine include increased urination as well as difficulty urinating; constipation as well as diarrhea; agitation and trouble sleeping but also drowsiness and unresponsiveness (Cymbalta no date).

So the term *side effect* is subtly and dangerously misleading. It implies atypical, not important, as well as unintended. But a drug, a material chemical substance, just does what it does, whether we want it to or not. When the typically short clinical trials turn up only a few unwanted effects in only a few people, that does not mean that other people are not experiencing the same effects in latent, not-yet-observable form. We should rather expect that under sufficiently long treatment, many, most, or even all patients would experience those unwanted "side" effects to an appreciable extent. Drugs prescribed for a chronic condition—blood thinners, pressure-lowering drugs, statins, anti-diabetes drugs—are to be taken forever, so any possible side effects are quite likely to show up sooner or later in quite a lot of people.

Current practice came about because reliance on surrogate markers supplanted feelings of illness on the part of patients and subsequent clinical diagnosis by a physician (Greene 2007). Nowadays we're urged to "know our numbers"—cholesterol, blood sugar, blood pressure, and more. In earlier times, high blood pressure, "hypertension," was diagnosed because of such symptoms as severe headache, dizziness, blurred vision, chest pain. But nowadays hypertension is diagnosed on the basis of blood pressure alone, whether or not there are any symptoms—even though the Institute of Medicine (2010) has acknowledged that blood pressure is not a valid surrogate for cardiovascular disease.

About a century ago, after measuring blood pressure had become routine, it was soon found that it varies over a wide range among manifestly healthy people, and for any given individual it also varies with time of day, temperature, and most notably stress. An average rule of thumb used to be that blood pressure approximates age plus 100 (Graveline n.d.). But even though the normal, natural increase with age is well-known, the official designation of hypertension is independent of age (PubMed Health 2011)!

> Normal blood pressure is . . . lower than 120/80 mmHg most of the time.
> High blood pressure (hypertension) is . . . 140/90 mmHg or above most of the time.
> If . . . 120/80 or higher, but below 140/90, it is called pre-hypertension. If you have pre-hypertension, you are more likely to develop high blood pressure.

Well, of course you are. If your pressure is between 120 and 140, all you have to do is to live sufficiently longer and it will come to exceed 140.

Knowing that blood pressure increases normally with age, it would be rational to define as too high—hypertension—pressures that exceed by (say) 50% what is average *for a given age*. Yet current practice recommends that everyone should have blood pressure lowered to about what is average for people in early middle age; and even lower if you have a condition that is not even known to be caused by high blood pressure (PubMed Health 2011):

> If you have heart or kidney problems, or if you had a stroke, your doctor may want your blood pressure to be even lower than that of people who do not have these conditions.

Under the official definition, then, we all achieve hypertension by about 60 years of age. According to the Institute of Medicine, 75–80% of Americans above age 60 have hypertension. I suggest that it is absurd to regard and treat as sick, about one-third of American adults including 75–80% of seniors, none of whom may have any feeling of being ill. And furthermore, it seems dangerous to administer drugs to be taken lifelong that are intended to counteract this normal age-related increase in pressure. In fact, half a century ago when diuretics were first being marketed to reduce blood pressure, many cardiologists disapproved, calling it a dangerous experiment and pointing out that increasing pressure with age might well be a compensation for the decreased flexibility of arteries, so that more pressure is needed to ensure that enough blood reaches the extremities and all organs (Greene 2007:53 ff.).

To conclude: The present-day drug-centered medical paradigm misinterprets natural accompaniments of aging as illnesses and prescribes lifelong medication with the real risk of debilitating so-called "side" effects. By contrast, an insightful geriatrician has pointed out that the challenges of aging are spiritual rather than medical, and that over-testing and over-treating, merely inconvenient in middle age, border on assault when perpetrated on older people, in whom almost every test is likely to reveal something "out of the ordinary" (Goodwin 1999). Furthermore, since clinical trials rarely if ever enroll people appreciably older than middle age, even the theoretical actual efficacy of drugs taken for chronic conditions is not really known. Enough is known, though, to indicate that older individuals should be cautious about taking even some quite commonly used medications (Beers no date).

Not only chronic ailments are being mis-treated with drugs. Emotional or mental conditions are being treated by drugs that have not been shown to do what they claim to do. Instead, there are nowadays far more Americans chronically disabled and dependent on drugs than were ever in asylums before treatment by drugs superseded other treatment of psychiatric conditions (Whitaker 2010).

It is impossible to estimate how much harm is being done by the paradigm of drug-based treatment of chronic ailments associated with aging. More than a century ago, the Food and Drug Administration was established as a counterweight to the prevalent peddling of panaceas and elixirs of life by confidence men, "snake-oil salesmen." The current practice of promiscuous prescribing of drugs makes it seem as though medicine has regressed by a century or so to those days of snake-oil salesmen—except that nowadays the peddlers are pharmaceutical corporations and not individuals.

But then we have learned recently from the Supreme Court that corporations are really people, so perhaps nothing at all has really changed.

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