

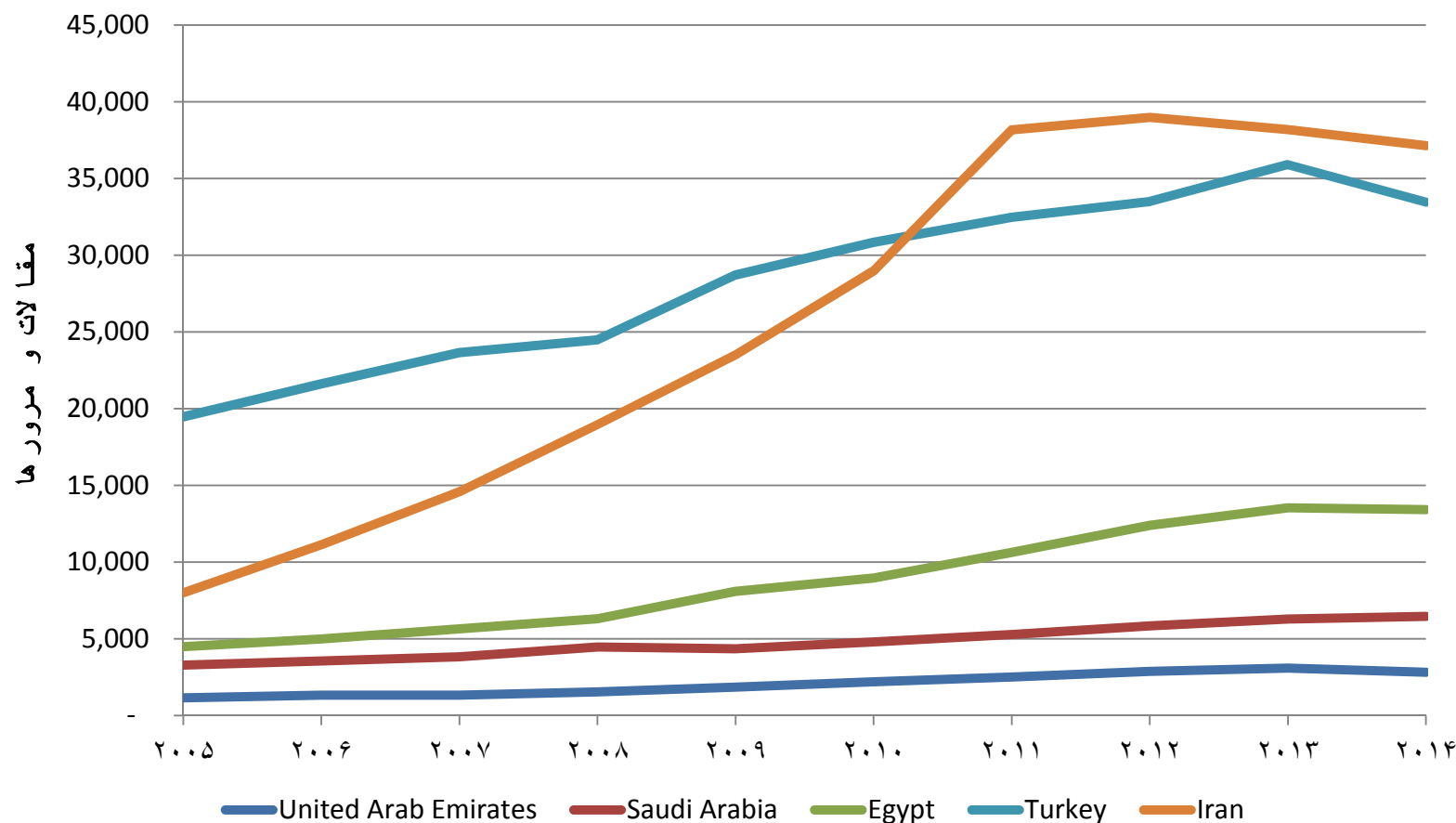
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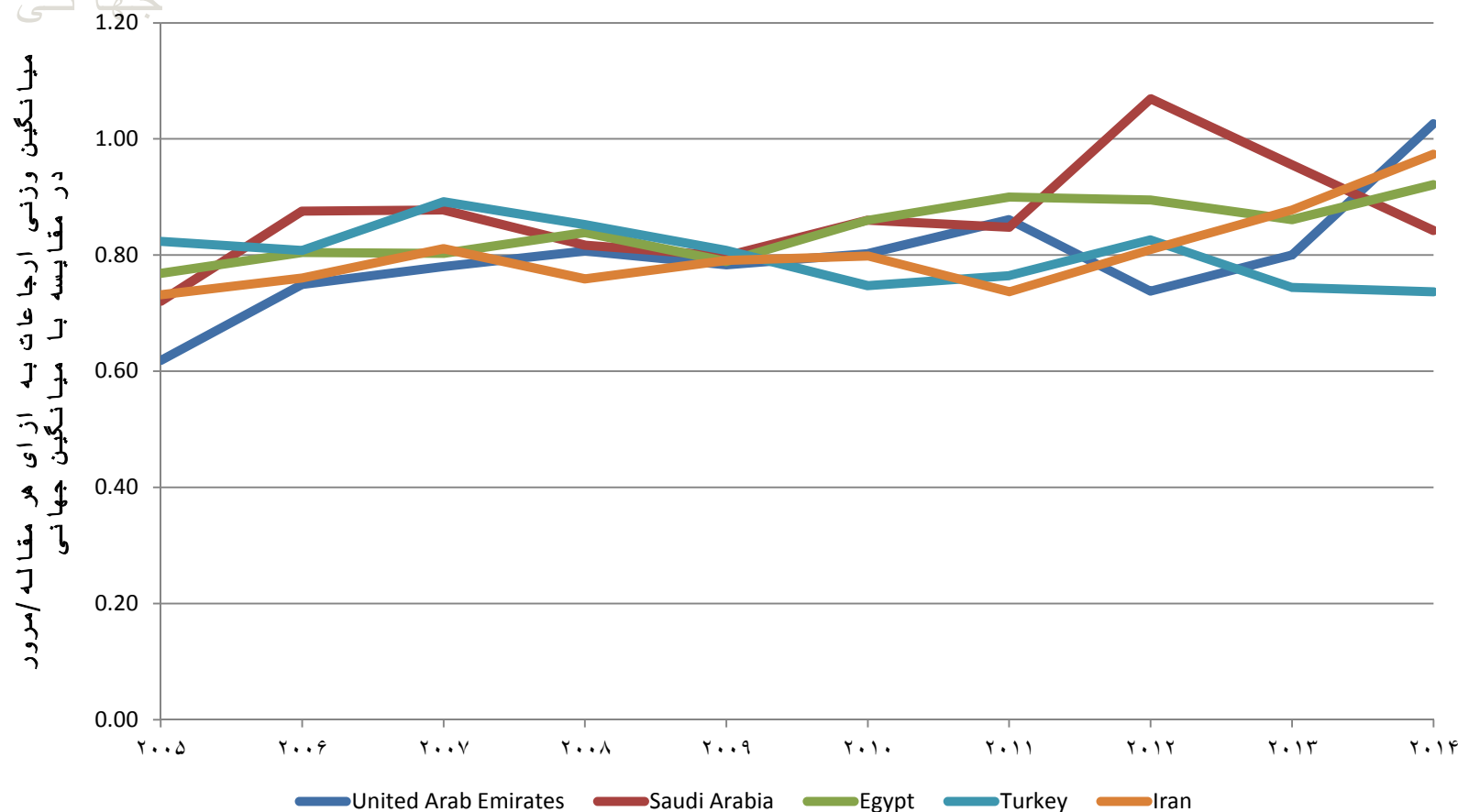
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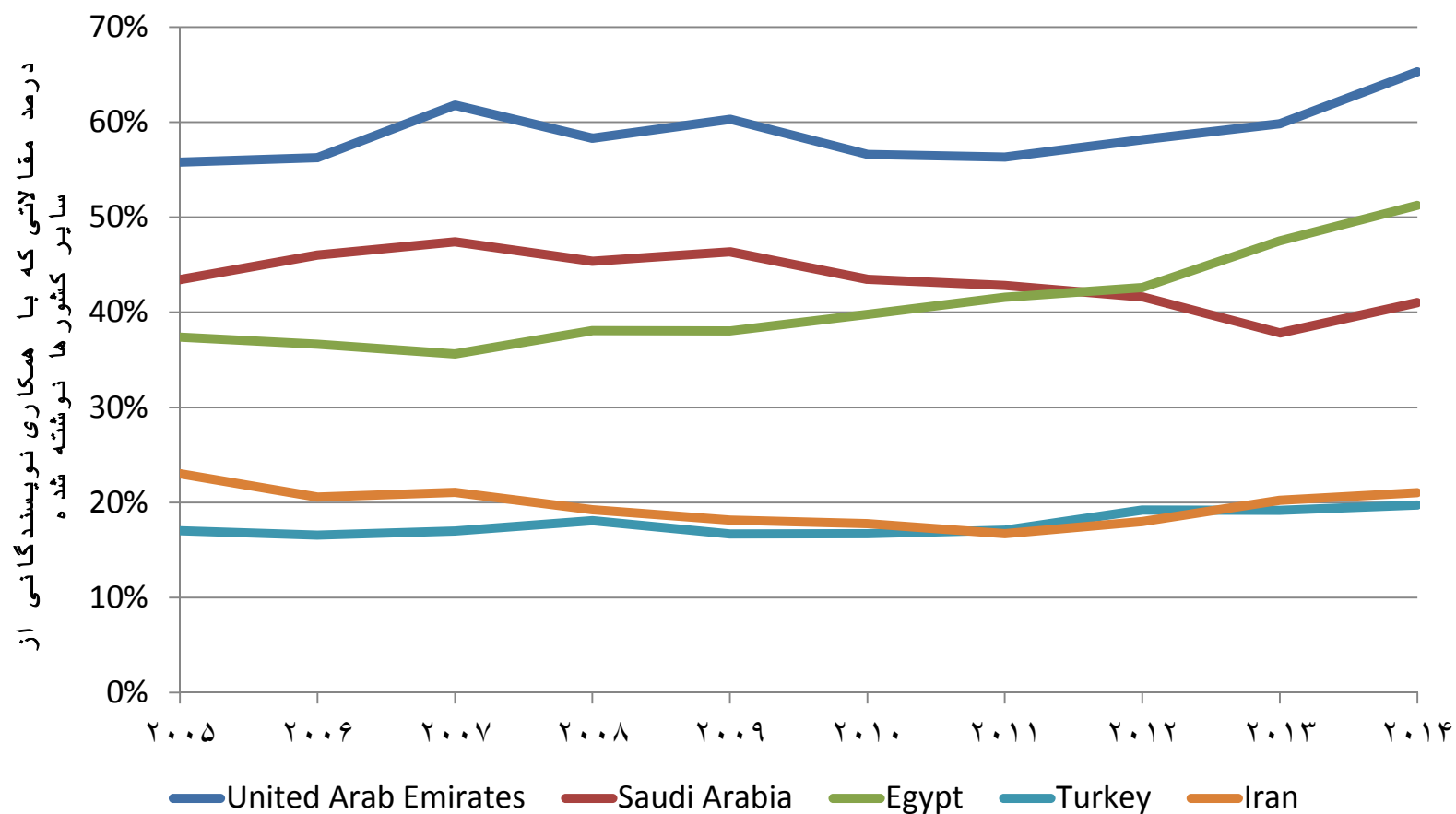
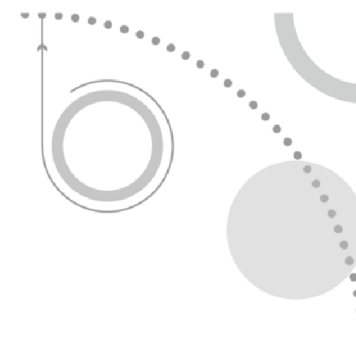
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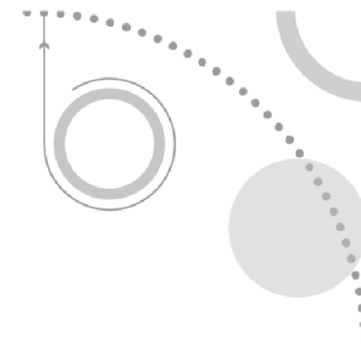
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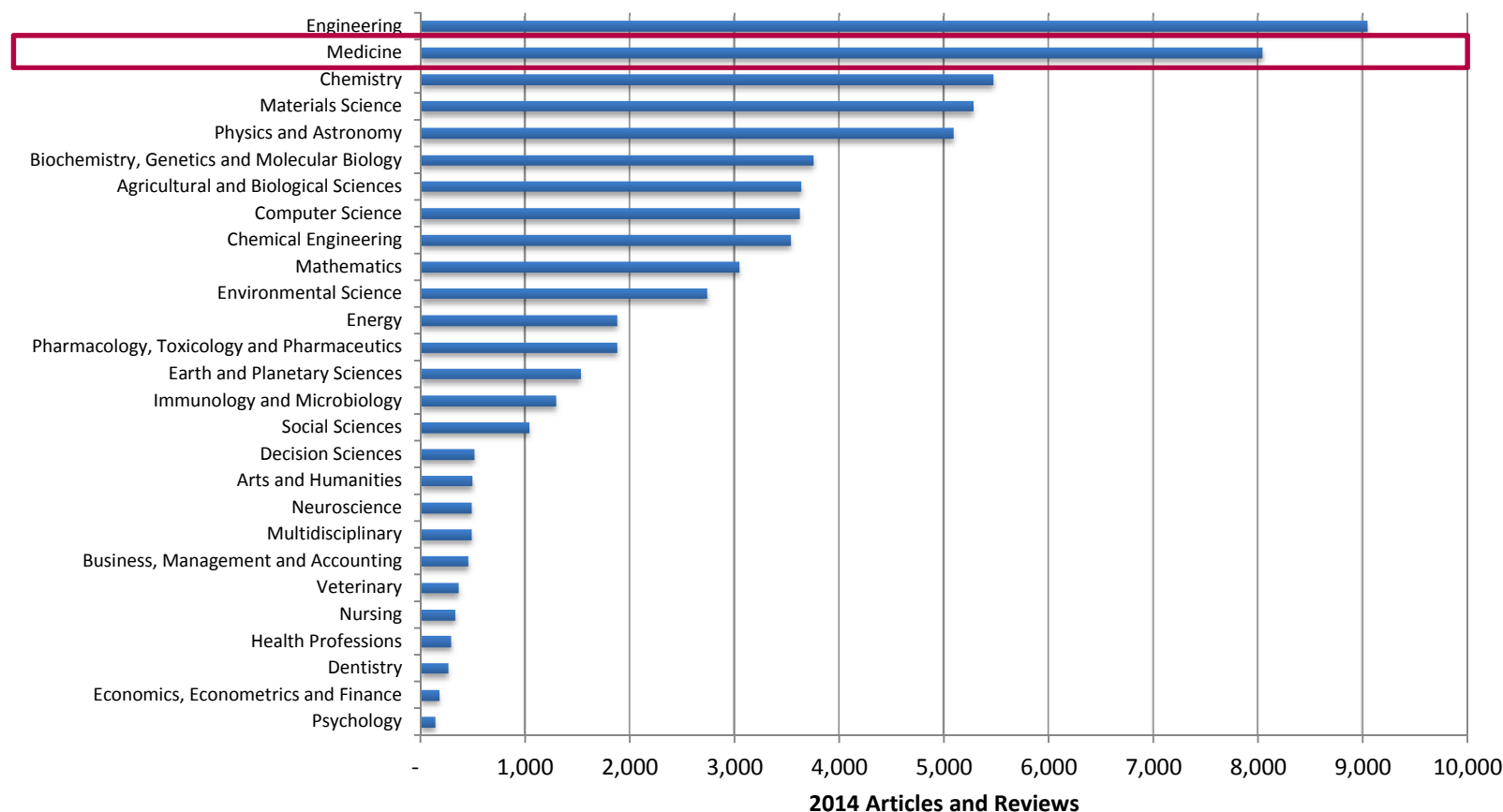
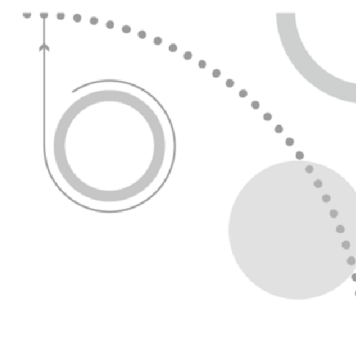
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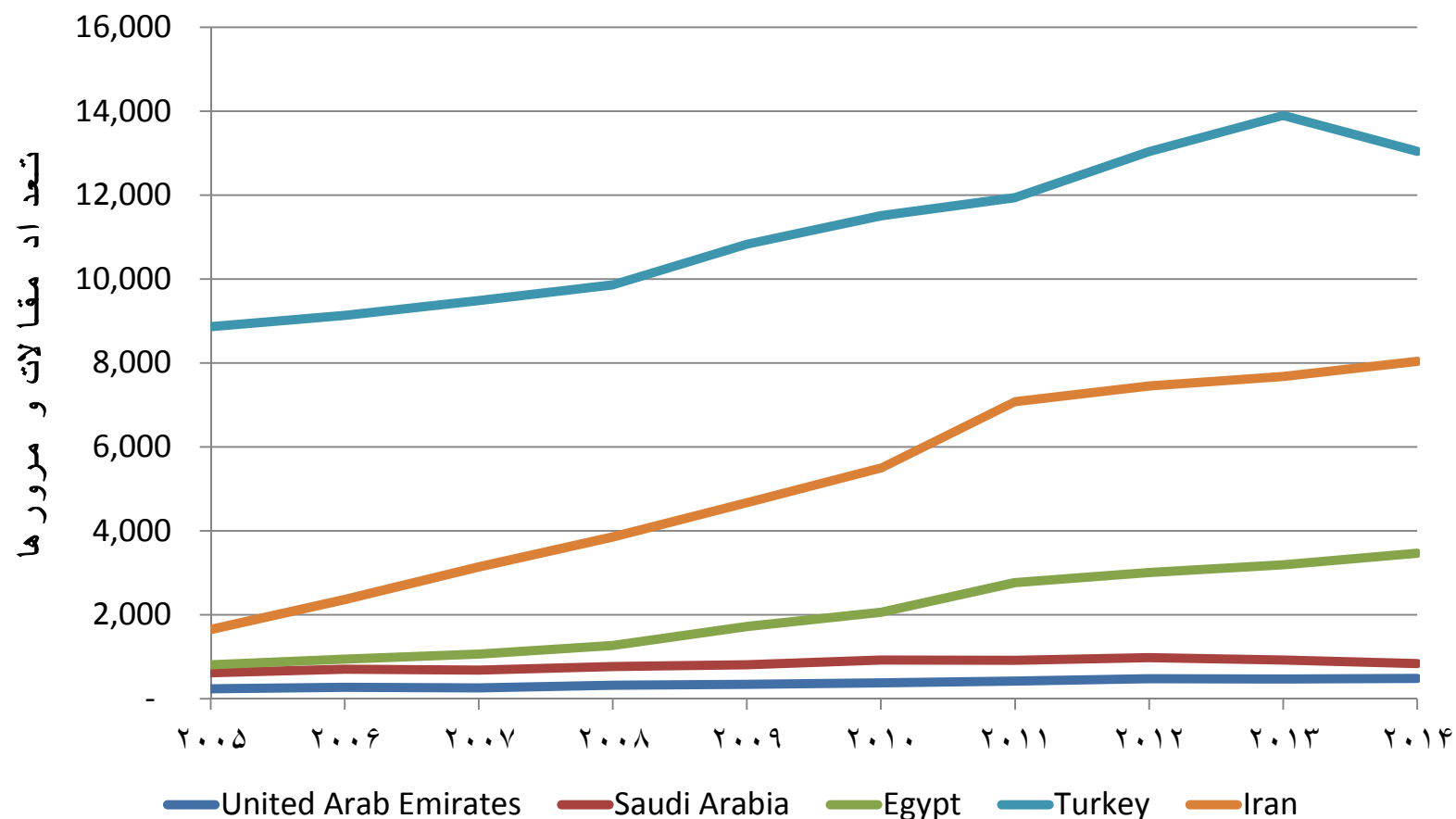
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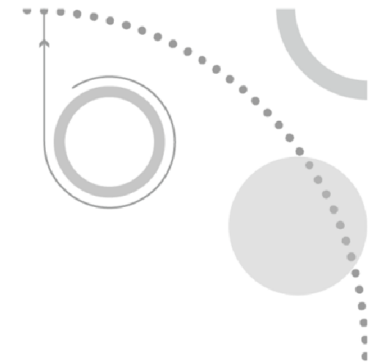


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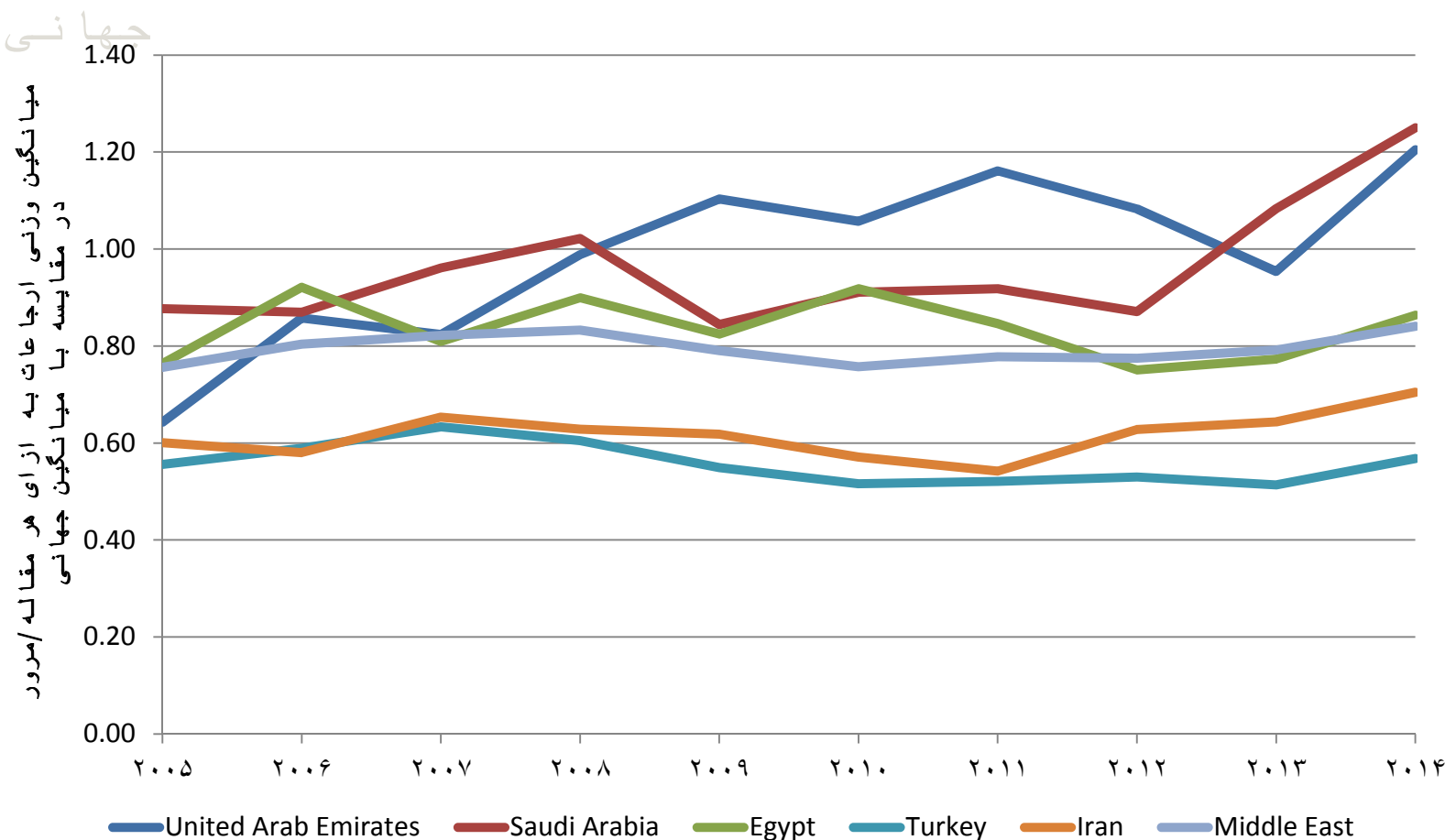
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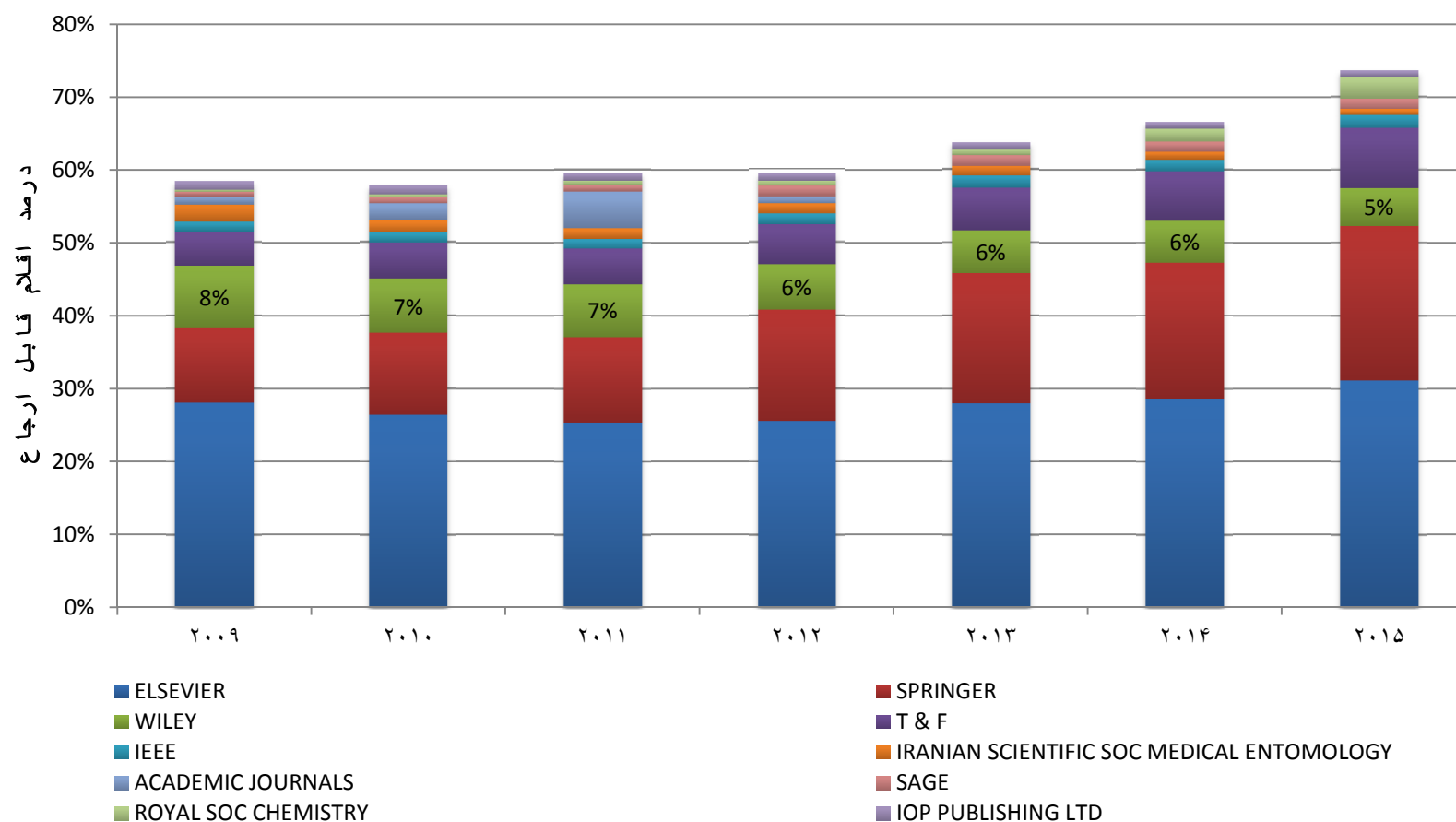
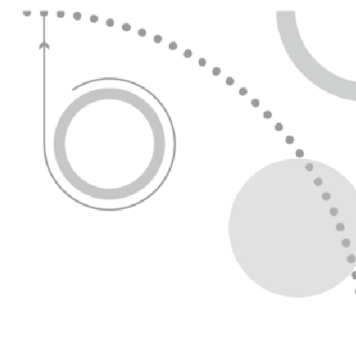
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Iran	17%
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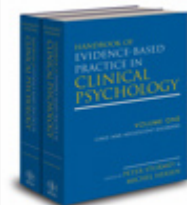
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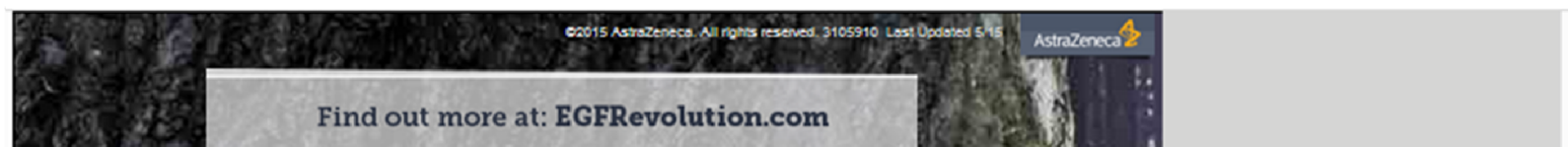
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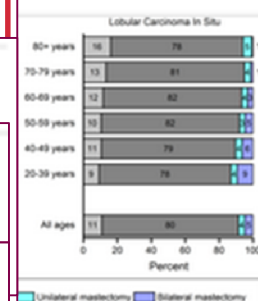
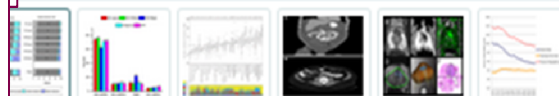


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Advances in diagnosis and treatment of cancer have greatly improved survival. With reduced mortality, morbidity related to cancer and its treatment has garnered increased attention, and issues surrounding quality of life have become ever more important. Cancer survivors have long reported cognitive dysfunction at various stages of the disease course with associated consequences upon well-being and functional independence. Nonetheless, until relatively recently, cancer-related cognitive impairment (CRCI) in patients with noncentral nervous system (non-CNS) malignancies was largely unacknowledged.^[1] The prevailing attitude was reinforced by the belief that chemotherapies were unable to cross the blood-brain barrier,^[2-4] precluding the possibility of a direct neurotoxic effect of cancer therapies. However, since the 1990s, a growing body of literature has verified the existence of CRCI, with recent animal models and neuroimaging studies uncovering pathophysiologic correlates.^[4-14]

CRCI research has largely focused on neurotoxicity associated with chemotherapy, often referred to as "chemobrain" or "chemofog."^[5] However, CRCI has also been documented in the absence of chemotherapy, leading to hypothesized associations with cancer itself,^[15] surgery,^[15-18] and other adjuvant therapies.^[19-21] Estimates of the prevalence of CRCI vary widely, although current longitudinal studies suggest that approximately 40% of cancer patients have evidence of CRCI before any treatment, up to 75% may have cognitive decline during treatment, and up to 60% exhibit deterioration in cognition even after completion of therapies.^[5, 22-25] The pattern of CRCI differs across patients and disease course, although severity is typically mild to moderate in nature. Mild cognitive impairments are conventionally considered to be performances that are from -1.5 to -2 standard deviations below population normative means. However, mild to moderate CRCI may also refer to a psychometrically significant decline relative to a patient's own pretreatment baseline performance (ie, a decline of approximately 1-2 normative standard deviations from baseline scores). Accordingly, these methods of determining impairment yield some difference in absolute impairment levels, depending on the individual patient's premorbid level of function. Nonetheless, the severity of CRCI is generally milder than the cognitive impairment typical of common neurologic populations, including those with neurodegenerative diseases and stroke. Despite this, encephalopathies involving dementia have been observed in the context of treatment with some cytostatic agents.^[26, 27] In addition, CRCI can persist for months to years after treatment,^[28] and even subtle impairments can have profound consequences upon quality of life, including occupational and social functioning.^[1]

To date, the majority of CRCI research in patients with non-CNS cancer has involved women with breast cancer,^[29, 30] who represent approximately 22% of the 14.5 million cancer survivors in the United States alone.^[31] Investigations have also been conducted in patients with testicular cancer,^[32, 33] lymphoma,^[28] multiple myeloma,^[34] colorectal cancer,^[35] ovarian cancer,^[36] and prostate cancer,^[37] among others. However, much of the literature regarding these populations is preliminary, with studies mostly consisting of small sample sizes. Future large, longitudinal, cohort studies are needed to better describe the prevalence and nature of CRCI in these patient populations. In light of the current state of the literature, this review deliberately focuses on the findings from clinical and basic research on CRCI in adult patients with breast cancer or preclinical models thereof, which has rapidly grown over the past few decades. To illustrate this work, the cognitive functioning of a hypothetical breast cancer patient is described at various stages of her disease and treatment course, based upon published reports and our collective clinical experience. These clinical descriptions are supplemented by summaries of associated empirical findings and discussion of pathophysiologic underpinnings and potential intervention strategies.

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